The American Journal of Surgery

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June 1948

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SUTURES IN ANCIENT SURGERY

Celsus

(25 B.C.—40 A.D.)

ALTHOUGH not a medical man, it was Celsus who compiled the earliest known scientific medical work in Latin. *De Re Medica,* assembled largely from Greek sources, is regarded as one of the best textbooks of the period. Two parts of its eight divisions are concerned with surgery. The most remarkable operation described by Celsus is the excision of the thyroid gland for the treatment of goitre. He also describes plastic operations on the face and mouth and technics adopted for the removal of tonsils and for penetrating wounds of the abdomen. Reference is made to the use of metallic sutures by surgeons of that early time who used "gold and iron" threads. For nearly a hundred years after Celsus' death, no Latin surgical works of importance are known to have been written.

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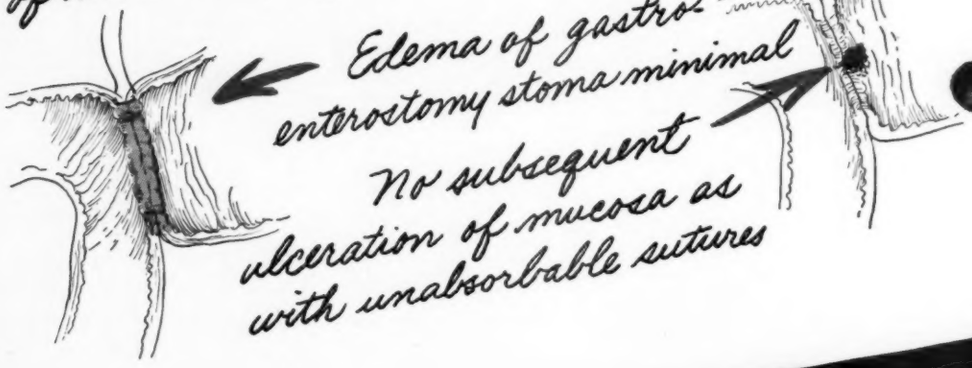

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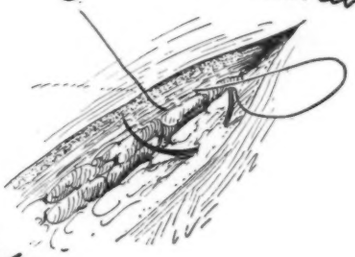

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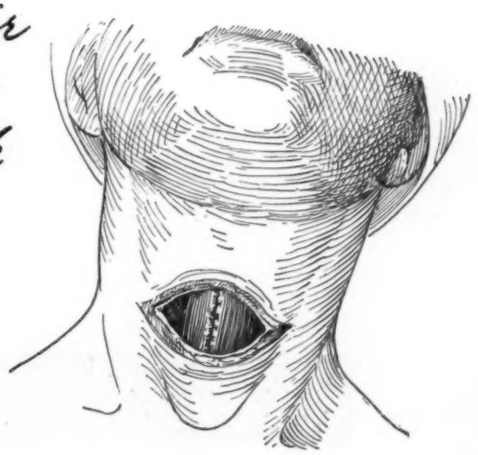

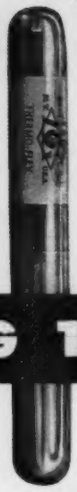

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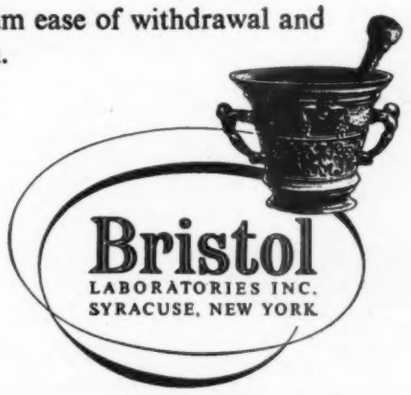

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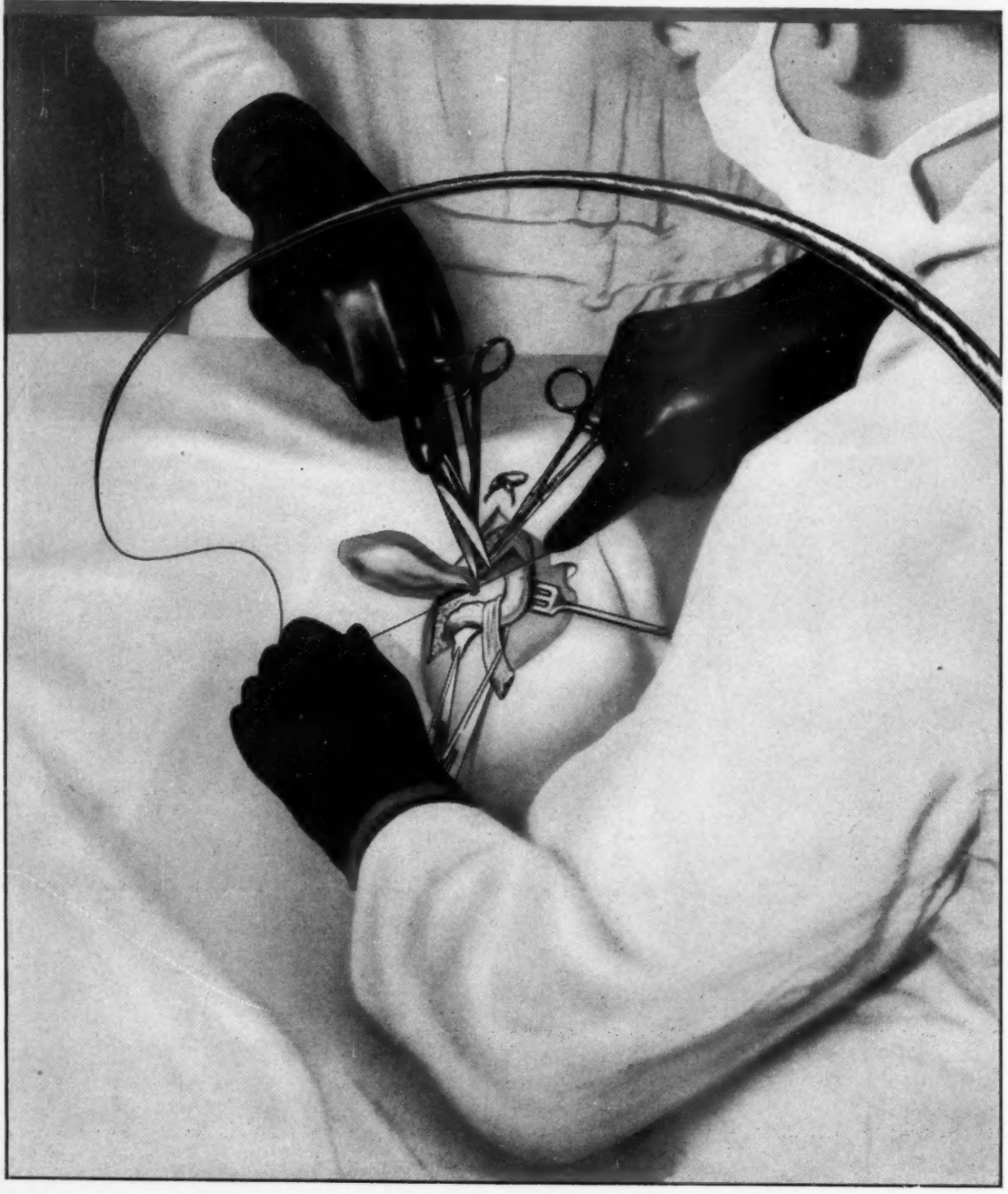

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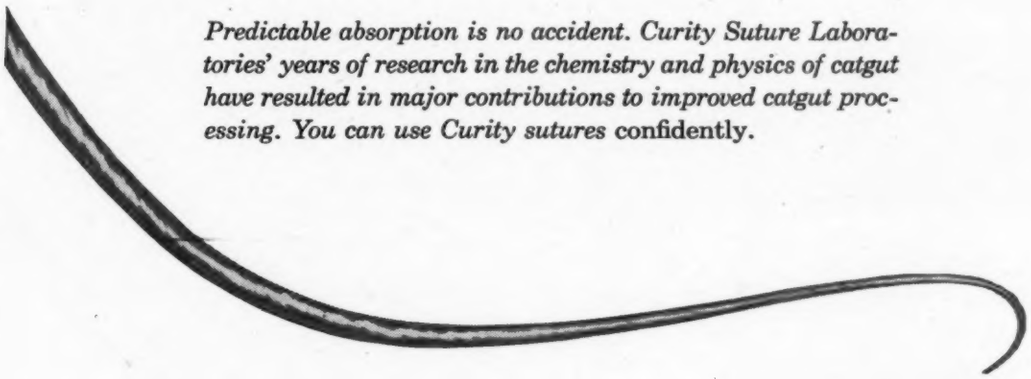

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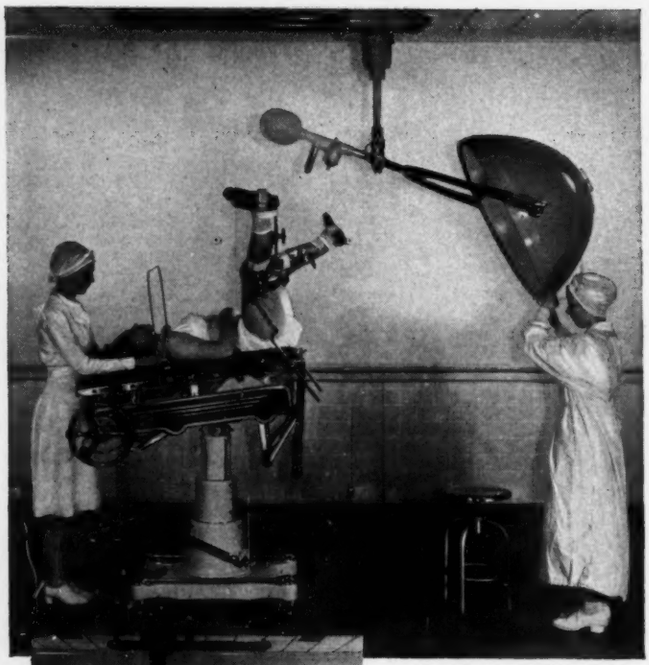

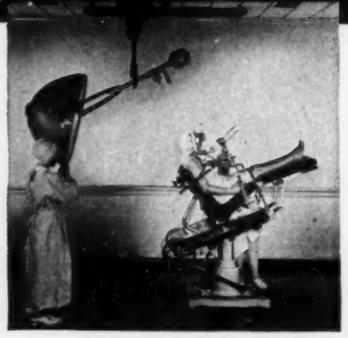

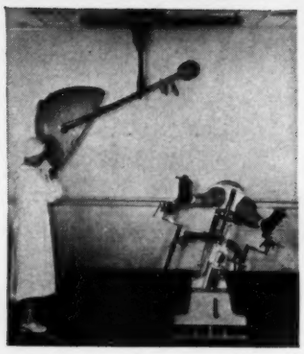

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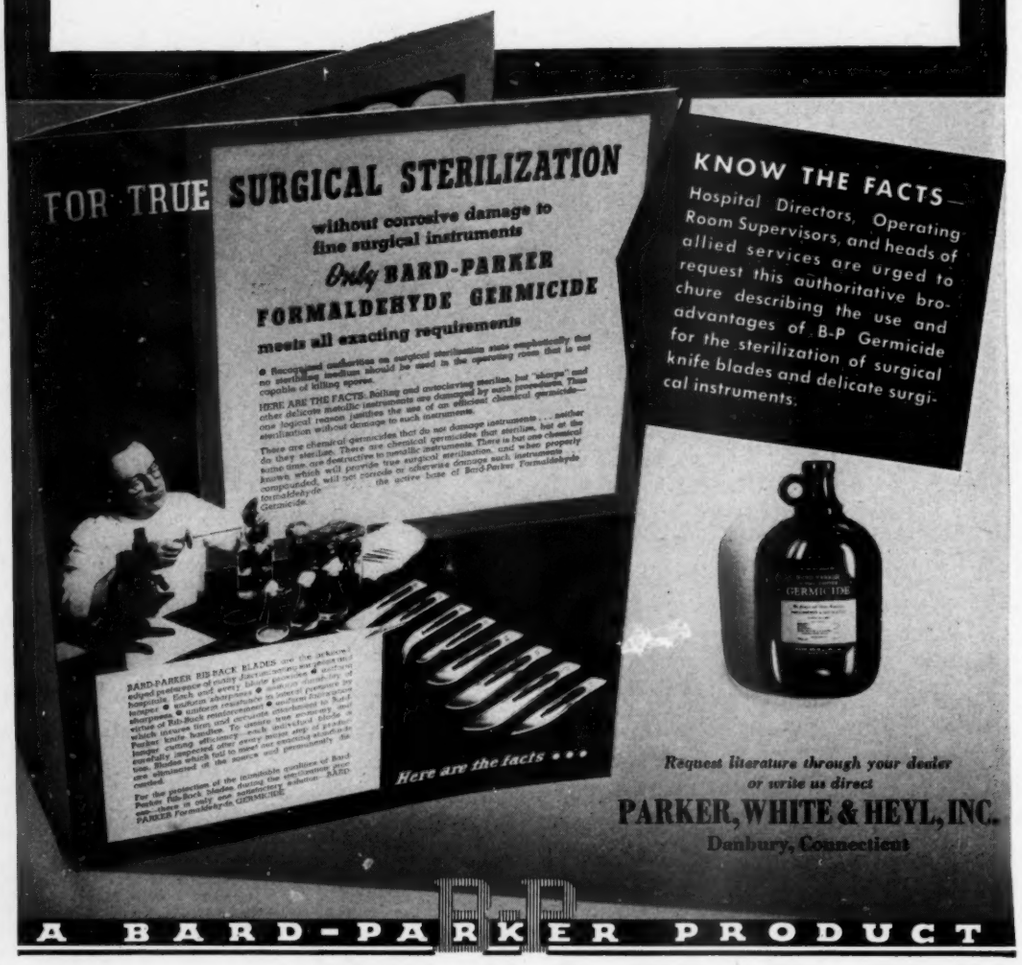
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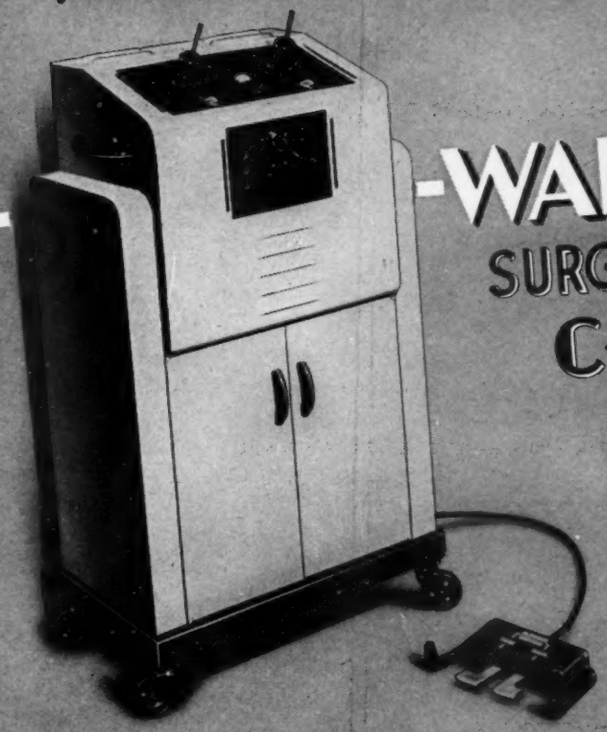

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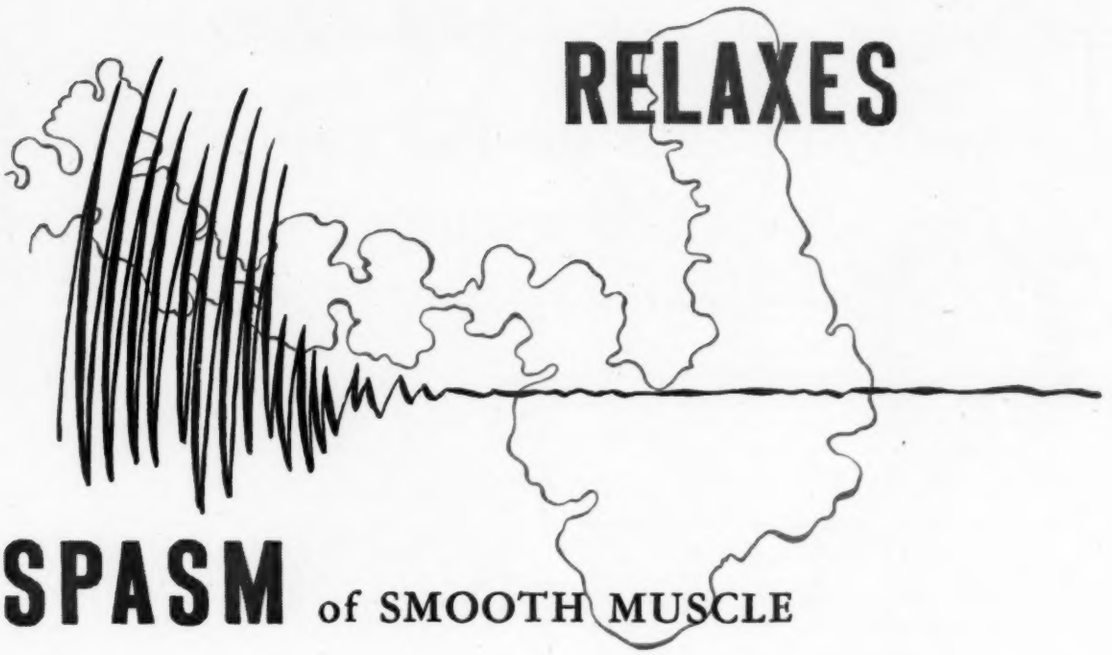

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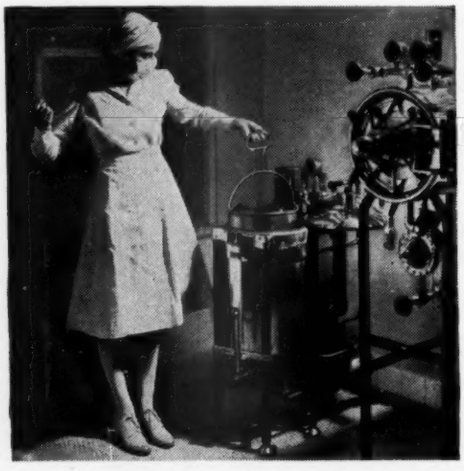

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(1533—M—4/5/1946)

Lipiodol*

completes the diagnostic picture!

Alteration in function as well as structure is graphically portrayed in X-ray studies of the accessory nasal sinuses with Lipiodol. Sharp contrast gives excellent structural outline. Displacement and emptying characteristics readily determine functional aberrations, thus completing the diagnostic picture.

In addition, its usefulness in bronchography, utero-salpingography and sialography, further establishes Lipiodol as an important adjunct in diagnostic roentgenography.

A contrast medium of choice because . . .

1. Firmly-bound 40% iodine in poppyseed oil insures uniform radiopacity.
2. Its viscosity produces clear delineation without excessive "pooling".
3. Exclusive formulation does not involve use of chlorine or its derivatives.
4. Bland Lipiodol insures minimal irritation to mucous membranes.

Roentgenogram with Lipiodol
(1533—M—4/15/1946)

Lipiodol is the registered trade-mark for the original product created by Lafay. This product alone can bear the name Lipiodol. Made in the U. S. A. Available in a variety of ampule sizes and 20 cc. multiple dose vials.

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- **stable in storage**
- **easily administered**
- **quantitatively absorbed**

high potency
vitamin B complex
& ascorbic acid

LYO B-C Principal B-Complex Factors and Ascorbic Acid provides an accurate and positive means of treatment with the essential, water-soluble vitamins. • Preserved by the lyophile technic, indefinitely stable without refrigeration, and conveniently administered by intramuscular or intravenous injection, or addition to intravenous infusions, LYO B-C Vitamins assure total absorption of vitamins B_1, B_2 (G), B_6, C, calcium pantothenate, and niacinamide. The dose is received quantitatively, thus avoiding the uncertainty and inefficiency of enteric absorption. • LYO B-C Vitamins are indicated for high potency treatment of water-soluble vitamin deficiencies encountered in surgery and in medical practice. Sharp & Dohme, Philadelphia 1, Pa.

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Each 5-cc. 'VACULE' vial contains:

Thiamine hydrochloride (vitamin B_1)	100 mg.
Riboflavin (vitamin B_2)	10 mg.
Pyridoxine hydrochloride (vitamin B_6)	10 mg.
Calcium pantothenate	50 mg.
Niacinamide	250 mg.
Ascorbic acid (vitamin C)	200 mg.

LYO B-C®

Principal B-Complex Factors and Ascorbic Acid
For Parenteral Use

Outside the Ivory Tower

A NEW book on a much neglected subject is both a joy and doubly welcome. Such a work has been published by The Macmillan Company. It is entitled "The Aseptic Treatment of Wounds" by Carl W. Walter, Assistant Professor of Surgery, Harvard University, and the illustrations were done by Mildred B. Codding, Surgical Artist, Peter Bent Brigham Hospital. This monograph is an attempt, and a successful one, to correlate the knowledge and effort of all who contribute toward the aseptic treatment of wounds.

It was written primarily to serve as a text for medical school courses, but we are sure interns, surgical residents and trained surgeons will find a copy of this book a valuable possession; and we strongly urge every one, physician and nurse, who is connected in any manner with the surgical domain to own and carefully read this remarkable work. Although the technic described is that elaborated at the Peter Bent Brigham Hospital and expresses the surgical philosophy of Harvey Cushing and Elliott C. Cutler, any one employing one of several other accepted technics will study this book with profit.

Some of the chapter headings are: The Importance of the Aseptic Treatment of Wounds, The Development of the Concept of Sepsis, Chemical Destruction (Physical Instruction in another chapter) of Bacteria, Chemical Disinfection of Instruments, Sterilization by Steam, Sterilization by Dry Heat, Disinfection of the Skin, Operating Room Technic, Preparation of Parenteral Fluids, Hospital Infection of Wounds, Maintenance of Sterilizing Equipment, etc. There is an ample Index. Elliott C. Cutler says in the Foreword, "This is but a small volume (372 pages) and its contents should be known to everyone who takes upon his shoulders the responsibility of opening the human body. There can be little excuse now for sepsis following an operation of election, since the methods of avoiding it are presented here in simple words and with complete explanation. . . . " The illustrations are superbly done. Truly this is a book marked a "must."

INDISCRIMINATE substitution of practical nurses for registered professional nurses to take over the major part of ordinary bedside nursing, as suggested by Dr. Morris Fishbein of the American Medical Association, was assailed by Ella Best of the American Nurses' Association as "detrimental and dangerous" to a patient's health. Her opposition to Dr. Fishbein's views, which appear as an editorial in the health magazine, Hygeia, was made known in a telegram sent to Dr. Fishbein: "May we respectfully suggest that the solution of the nursing crisis does not lie in the training of more practical nurses to take over the major part of bedside nursing in hospitals, as you were quoted by the Associated Press with a Chicago dateline as saying in Hygeia. We recognize that practical nurses fulfill a needed function but not this one. This step would be extremely detrimental and dangerous to a patient's welfare," Miss Best wired. "In time of critical shortage of doctors it was not suggested that first aid technicians be substituted in their place. The same parallel exists between practical and professional nurses. . . .

CIBA Pharmaceutical Products, Inc., have brought out a new book of over 300 pages entitled, "Refresher Course in Male Hormone Therapy." It contains abstracts of the literature on this subject over a four-year period. All material has been taken from accepted medical publications. The book has been compiled to provide physicians with a comprehensive reference to the experimental work performed as well as the therapeutic uses of the male sex hormone. This well prepared book is made available to the profession without charge and will be sent to any physician requesting it.

(*continued on page* 36)

Relief Beyond Belief

Ultracain Ointment's soothing anesthetic action is due to the synergistic effect of its aminobenzoate esters. Even on repeated use, Ultracain Ointment applications are signally free from evidences of irritation.[1] This soothing quality—plus the pronounced bacteriostatic, fungistatic, and healing action—makes Ultracain Ointment of outstanding value in pruritus, hemorrhoids, fissures, cutaneous ulcers, burns, non-specific dermatitis, etc. Available in one ounce tubes with applicator and one pound jars.

[1] Combes, F. C. et al.: Am. J. Surg., 74:45, 1947

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MEAT
And The Surgical Patient

New knowledge regarding the significance of protein imbalance in the presence of surgical affections has profoundly influenced the management of the surgical patient.

Many conditions calling for surgery, such as peptic ulcer, cancer of the gastrointestinal tract, affections of the biliary tree, or bowel obstruction may lead to protein imbalance. Severe burns and major traumas usually are associated with considerable protein loss. The operative procedure, intended to correct the situation, initially only adds to the protein drain.

The protein-deficient patient is always a poor surgical risk, predisposed to shock and more vulnerable to infection and other complications. Gastrointestinal anastomoses, for instance, may fail to function properly because of edematous occlusion of the stoma. Healing may be seriously impaired and convalescence delayed.

To make the patient a better risk, and to accelerate recovery, diets high in biologically complete protein are now routinely instituted before elective surgery is undertaken. Convalescence appears to progress better when a high protein diet is speedily resumed postoperatively.

Protein losses or deficiencies should be corrected by oral feeding of natural foods whenever feasible.

Meat, an excellent, highly digestible source of biologically complete protein, is particularly desirable in the patient's diet. It is usually well tolerated before surgery and can be given after surgery as soon as solid foods are permitted.

The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

American Meat Institute
Main Office, Chicago...Members Throughout the United States

44...

of Nembutal's Clinical Uses

SEDATIVE

Cardiovascular
Hypertension[1]
Coronary disease[1]
Angina[1]
Decompensation
Peripheral vascular disease

Endocrine Disturbances
Hyperthyroid
Menopause—female, male

Nausea and Vomiting
Functional or organic disease (acute gastrointestinal and emotional)
X-ray sickness
Pregnancy
Motion sickness

Gastrointestinal Disorders
Cardiospasm[2]
Pylorospasm[2]
Spasm of biliary tract[2]
Spasm of colon[2]
Peptic ulcer[2]
Colitis[2]
Biliary dyskinesia

Allergic Disorders
Irritability
To combat stimulation of ephedrine alone, etc.[2,3]

Irritability Associated With Infections[4]

Restlessness and Irritability With Pain[5,6]

Central Nervous System
Paralysis agitans
Chorea
Hysteria
Delirium tremens
Mania

Anticonvulsant
Traumatic
Tetanus
Strychnine
Eclampsia
Status epilepticus
Anesthesia

HYPNOTIC
Induction of Sleep

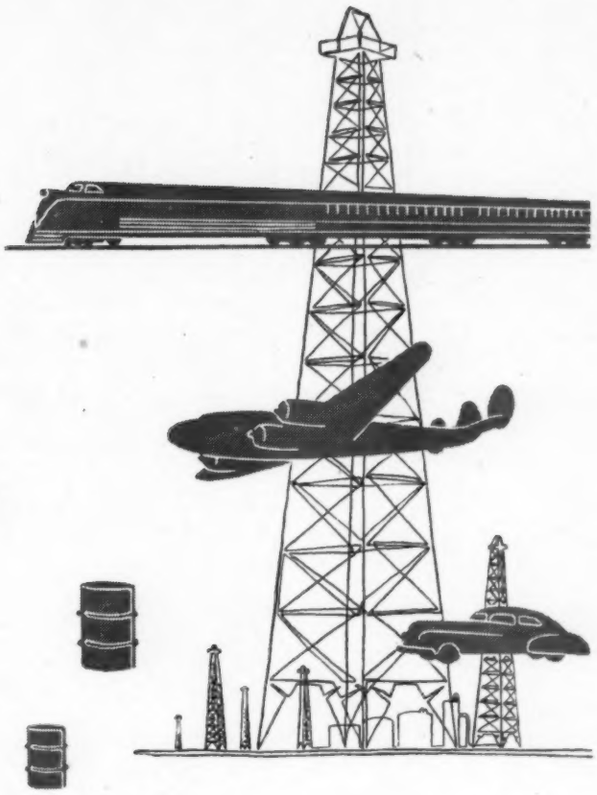
OBSTETRICAL
Nausea and Vomiting
Eclampsia
Amnesia and Analgesia[6]

SURGICAL
Preoperative Sedation
Basal Anesthesia
Postoperative Sedation

PEDIATRIC
Sedation for:
Special examinations
Blood transfusions
Administration of parenteral fluids
Reactions to immunization procedures
Minor surgery

Preoperative Sedation

Nembutal alone or [1]Glucophylline® and Nembutal, [2]Nembutal and Belladonna, [3]Ephedrine and Nembutal, [4]Nembudeine®, [5]Nembutal and Aspirin, [6]with scopolamine or other drugs.



a product put to many uses

Many and varied, too, are the uses of short-acting Nembutal—more than 44 uses so far—described in over 460 reports. And as the list has grown, more and more physicians have extended the use of Nembutal in their practice. It is logical procedure. Their experience has shown that adjusted doses can achieve any desired degree of cerebral depression, from mild sedation to deep hypnosis . . . that the dosage required is only about *one-half* that of many barbiturates. They have learned that, with this small dosage, there is less drug to be inactivated, reduced possibility of "hangover," shorter duration of effect, a wide margin of safety, and definite economy to patients. The list of indications at right is only partial. If it suggests more ways in which you can effectively use short-acting Nembutal—available in 11 product forms—won't you give it a trial in your next few cases? Write for a copy of the new tab-indexed booklet, "44 Clinical Uses for Nembutal," containing indications, dosages, contraindications. ABBOTT LABORATORIES, North Chicago, Illinois.

In equal oral doses, no other barbiturate combines **QUICKER, BRIEFER, MORE PROFOUND EFFECT** *than . . .*

Nembutal®

(PENTOBARBITAL, ABBOTT)

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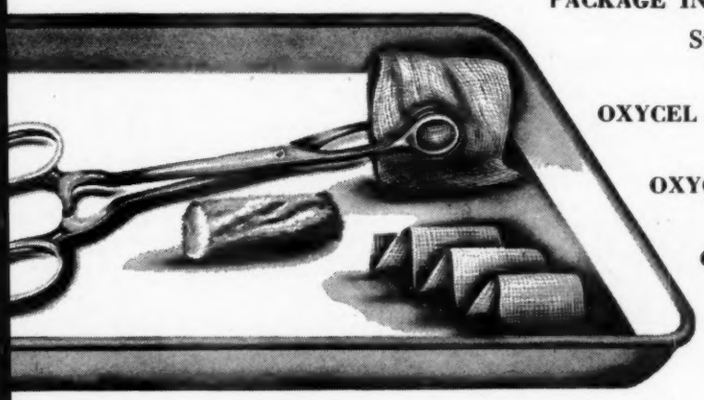

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OXYCEL PLEDGETS (Cotton-type)
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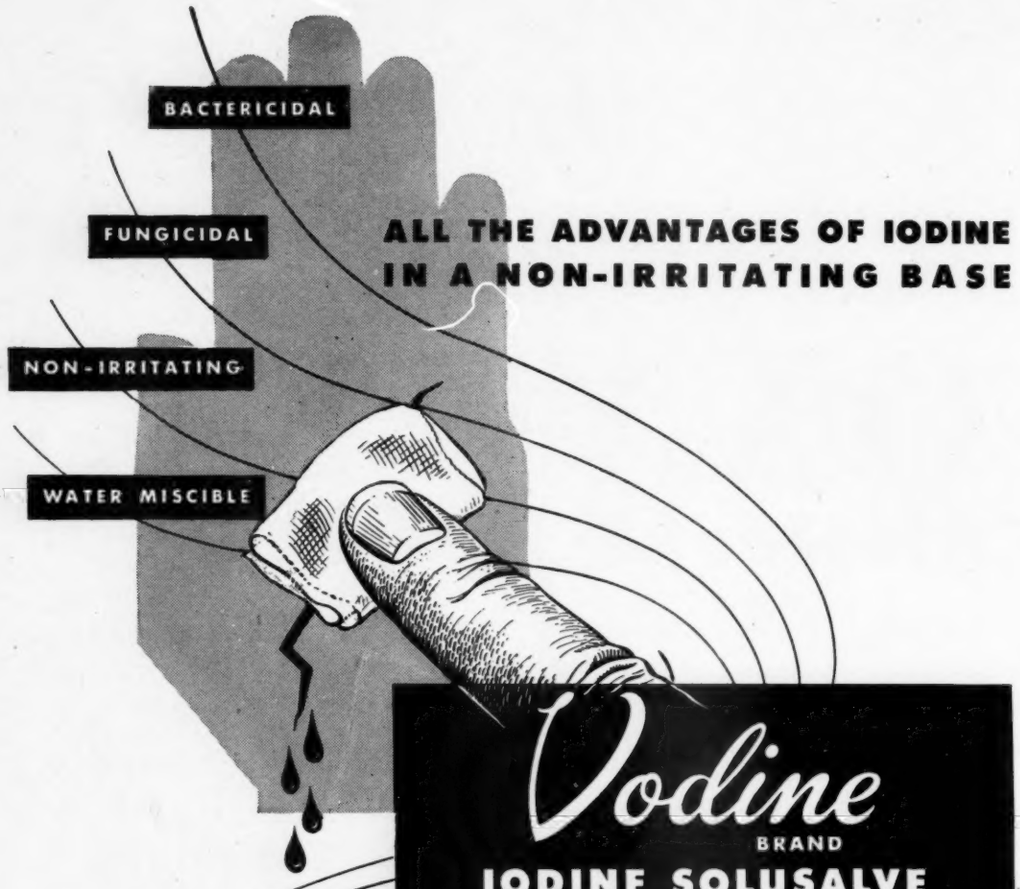

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Vodine
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For effective topical antisepsis without smarting, burning or staining, Vodine Brand Iodine Solusalve combines one of the strongest germicidal agents—iodine—with a bland, water-miscible, non-irritating base—solusalve.

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(ethinyl estradiol)

***"specially active . . . giving extraordinarily good results by oral administration . . ."*[1]**

Unrivaled potency permits *minute* dosage—*measured in hundredths of a milligram*—for rapid alleviation of menopausal distress and other estrogen-deficient states. Rarity of side reactions is noteworthy in therapeutic dosage. Promotion of a gratifying sense of well-being is a conspicuous feature of ESTINYL therapy. Low cost permits the prescription of ESTINYL* to any office patient for potent, highly effective, well-tolerated, oral estrogen therapy.

DOSAGE:
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ESTINYL (ethinyl estradiol) Tablets of 0.02 mg. (buff) or 0.05 mg. (pink), in bottles of 100, 250 and 1000. ESTINYL LIQUID, 0.03 mg. per 4 cc., in bottles of 4 and 16 oz.

1. Zondek, H.: The Diseases of The Endocrine Glands, ed. 4 (Second English), Baltimore, Williams & Wilkins Company, 1944, p. 421.

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AGAINST

PRE and POSTOPERATIVE

IRON-DEFICIENCY

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Iron-deficiency anemia is a factor to be considered in pre and postoperative conditions. Hence, we suggest that you prescribe Copperin routinely for a period preceding and following surgery. Copperin stimulates the hematopoietic system— provides material for replacement of hemoglobin—increases the red blood cell count.

Because in Copperin iron ammonium citrate is combined with the catalyst copper sulphate, there is a greater utilization of the iron by the system. For this reason the iron content per capsule is purposely small—32 Mgm. As there is no stomach irritation or constipating effect from Copperin, it is exceptionally well adapted to routine use. In two strengths: Copperin "A" for adults; Copperin "B" for children.

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GUSTATORY satisfaction, the subjective aim of eating, is not necessarily achieved by a nutritionally adequate meal. Even with good proportions and amounts of essential nutrients, other requisites must be met. Thus, even a well balanced meal may be drab and fail to produce the desired palatal satisfaction. In such instances, candy usually provides the needed finishing touch, and does much to dispel gustatory monotony. Not only children but also adults appreciate the feeling of satiety that candy brings at such times.

Because of impaired or jaded appetite, many hospitalized patients and particularly convalescents frequently get little pleasure out of a nutritionally adequate dietary. Candy will give such appetites a welcome fillip as well as make its own proportionate dietary contribution to the extent of its contained nutrients and its caloric value.

Candies made with milk, butter, eggs, fruits or nuts as well as pure carbohydrates present no difficulties in digestion, have few contraindications when a general diet is allowed, and often satisfy when no other component of the dietary can.

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● **He too** can *not* afford to be without the vitamin adequacy made simpler, more certain and more economical with a Unicap a day.

In the past 5 years 2.7c buys less and less food, less shelter and less clothes

BUT 2.7c buys more and more vitamins — all these

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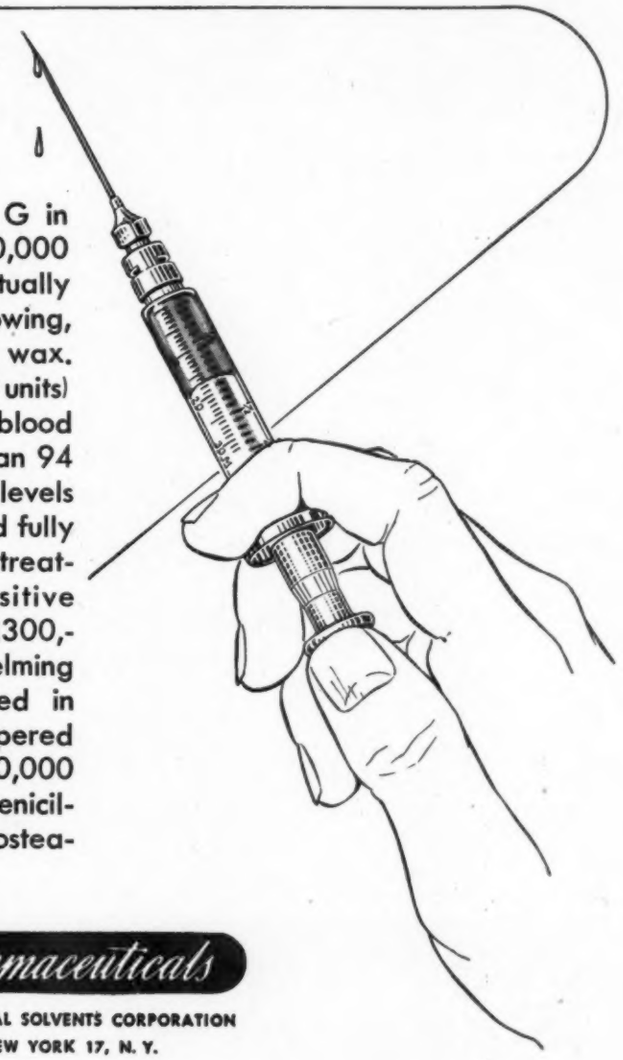

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FOR THE UTMOST IN PENICILLIN QUALITY . . . SPECIFY C.S.C. IN YOUR PRESCRIPTIONS

Only CHLOROPHYLL THERAPY

combines all these advantages:

Painful diabetic ulcer of about 4 months' duration which had not responded to ordinary treatment. Roentgenogram showed probable osteomyelitis. This was one of 50 cases of chronic ulcers described by Cady and Morgan in the April 1948 issue of *Am. J. of Surgery.*

- . . . accelerates healing
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- . . . reduces scar formation
- . . . nontoxic — bland and soothing
- . . . provides symptomatic relief
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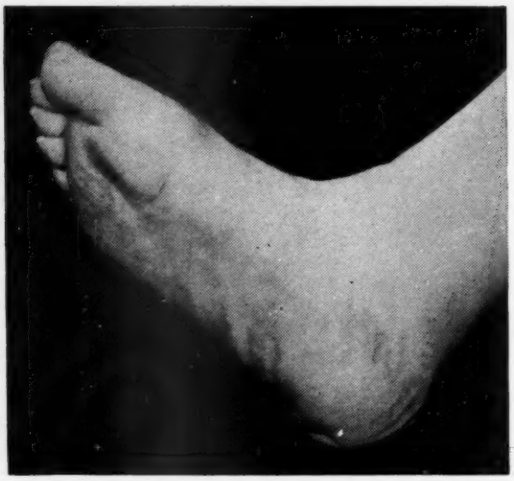

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Reports from clinicians emphasize the effectiveness of Chlorophyll Therapy as made possible by Chloresium

BOEHME, E. J.	The Treatment of Chronic Leg Ulcers with Special Reference to Chlorophyll	The Lahey Clinic Bulletin, 4: 242, 1946
BOWERS, WARNER F.	Chlorophyll in Wound Healing and Suppurative Disease	Amer. J. Surgery, LXXIII: 37 (1947)
CADY, J. B. and MORGAN, W. S.	The Treatment of Chronic Ulcers with Chlorophyll	Am. J. Surgery LXXV: 4 (1948)
GAHAN, E., KLINE, P.R. and FINKLE, T. H.	Chlorophyll in the Treatment of Ulcers	Arch. Dermat. & Syph. 47: 849 (1943)
HOLMES, G. W. and MUELLER, H. P.	Treatment of Post-Irradiation Erythema with Chlorophyll Ointment	Am. J. Roentgenol. 50: 210 (1943)
LANGLEY, W. D. and MORGAN, W. S.	Chlorophyll in the Treatment of Dermatoses	Penn. Med. Journal Vol. 51; No. 1 (1947)
MORGAN, W. S.	Chlorophyll Therapy A Review of 114 cases	Guthrie Clinic Bulletin, 16: 94 (1947)

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Dr. Lund served for many years on the faculty of Harvard Medical School and as surgeon-in-chief of Boston City Hospital. He served terms as president of the Clinical Congress of Surgeons of North America and the American Surgical Association.

From the Series, Great American Surgeons, Published by

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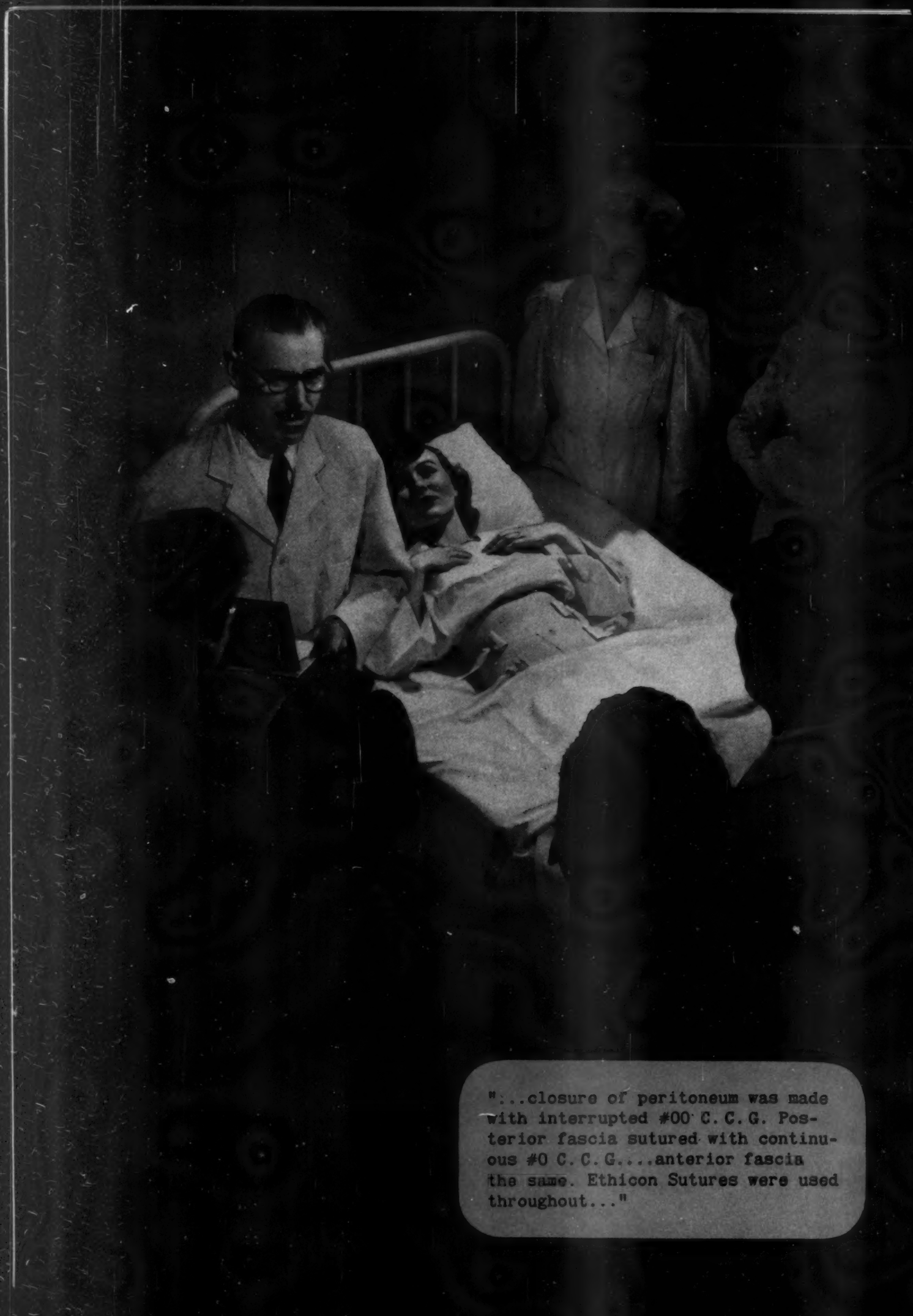

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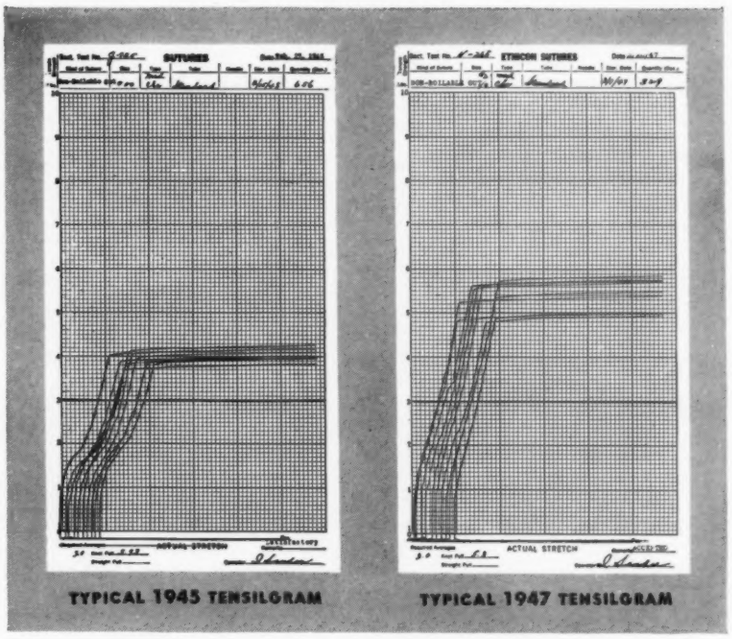

TYPICAL 1945 TENSILGRAM

TYPICAL 1947 TENSILGRAM

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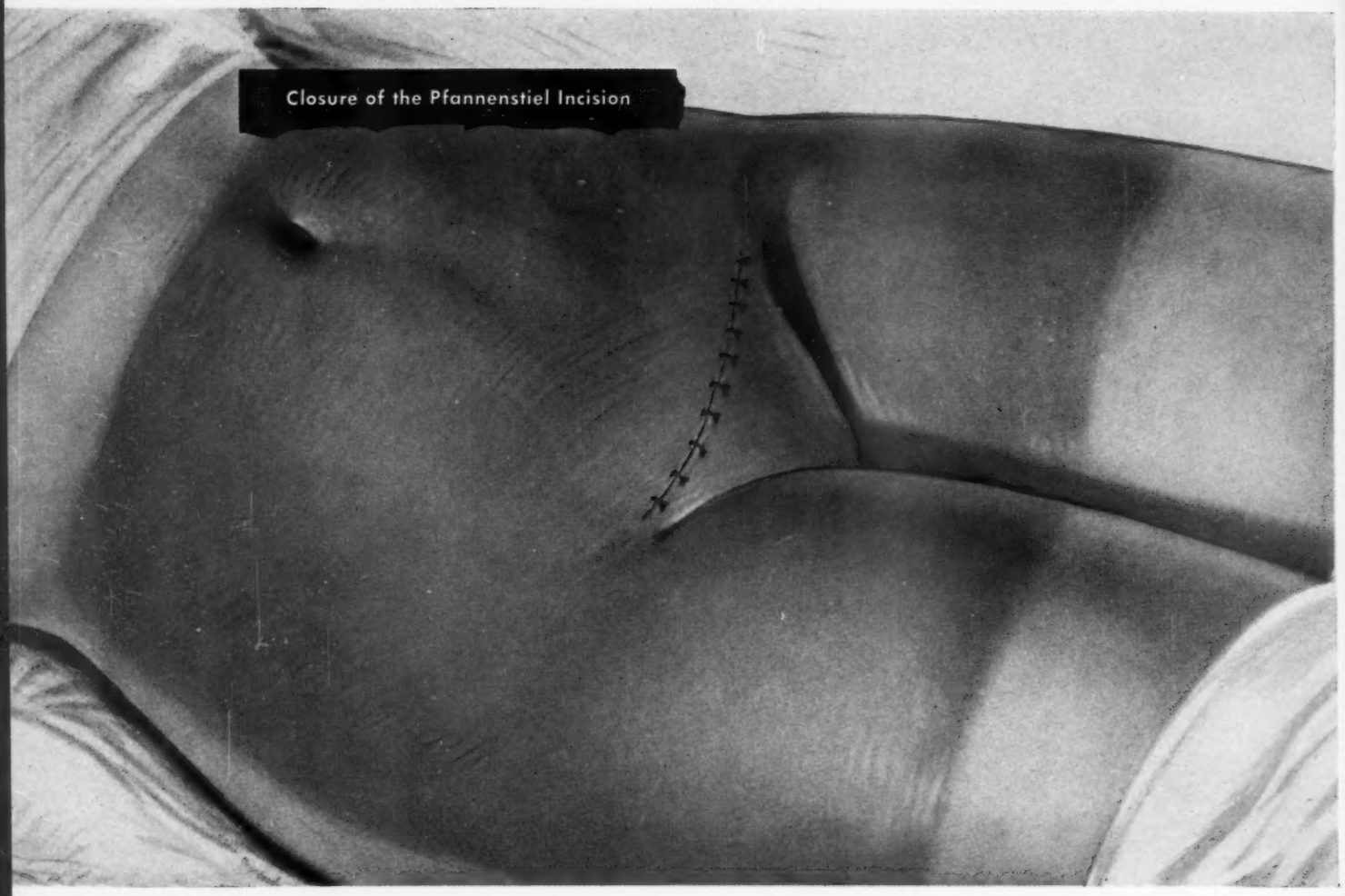

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Ethicon Silk forms smooth, firm knots. It has minimal adherence to tissue.

Ethicon Silk is non-capillary, serum-proof, non-toxic, and strictly U.S.P. gauge.

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Tensile strength is 10% to 30% greater than ordinary cotton. *Sizes are U.S.P.:* 4-0, 3-0, 2-0, 0. If you are a cotton man, try Ethicon Twisted Cotton.

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A PRACTICAL JOURNAL BUILT ON MERIT

Fifty-seventh Year of Publication

VOL. LXXV JUNE, 1948 NUMBER SIX

Editorials

SURGERY AND PSYCHOSOMATIC DISEASE

DURING World War II the medical profession became more acutely aware of psychosomatic disease than it had been for many years. However, this is a matter of terminology rather than actuality for the practitioners of medicine for generations have recognized that somatic pain may have its origin in psychic conflicts. That these psychosomatic phenomena may simulate surgical disease is not at all surprising when one recognizes the protean nature of the symptoms of so many of the surgical diseases and how few are the objective manifestations.

Abdominal diseases are expressed in the form of pain, alterations in the normal gastrointestinal and urinary responses such as anorexia, nausea and frequency of urination. The objective findings are also very few in the form of fever, tenderness and splinting of the musculature. With the possible exception of fever all of these may be present as expressions of psychosomatic disease. Fever may be present in minor degrees at the time of ovulation in the female and for a variety of minor causes including the psychic stress attending hospitalization. Selective dyspepsias may be an aid in the diagnosis of cholecystic disease yet this also is not infrequent in patients with psychosomatic disease.

How then may the organic disease be differentiated from the functional? Certainly all will agree that the operative process of elimination is neither the ideal nor even often warranted. This is the process of exploratory laparotomy, or the appendectomy or cholecystectomy for chronic but undiagnosed pain. Can the psychiatrist answer the question; can he differentiate the right lower quadrant pain of psychosomatic origin from that of the recurrent inflammations of the appendix? No! The psychiatrist can locate underlying causes for psychic conflicts within the patient but usually he requires that the clinician eliminate organic causes before he begins. Psychic conflicts of some kind are undoubtedly present in all humans to a greater or lesser degree and can be elicited by careful interrogation. That these are present does not mean that they are the cause of symptoms. One must beware of the adage *post hoc ergo propter hoc*. The psychosomatic patient may also have acute appendicitis, chronic cholecystitis, carcinoma of the pancreas and even carcinoma of the breast. Almost every patient who dies of carcinoma of the body or tail of the pancreas has been diagnosed as a psychoneurotic.

Wherein lies the answer to this double-edged sword of the term psychosomatic disease? Now, as it has for previous medical generations, it still remains a problem for the conscientious investigation by the

clinician, namely, careful analysis of the pain, its character, timing and radiation; a painstaking review of the complete anamnesis, again stressing the sequence or timing of the symptoms; a complete physical examination, utilizing all of the art of this procedure as distraction and false suggestion.

These, coupled with all of the refinements of scientific adjuncts, laboratory tests and radiologic studies, will bring to light most of the organic disorders. Discussion of the special problems with colleagues in the fields of internal medicine will further aid in the winnowing of the organic from the functional.

Yet, when all means available for study have been exhausted, there will still be patients in whom a failure of diagnosis must be admitted. Is the surgeon justified in considering his exploratory laparotomy a diagnostic procedure? This is an especially difficult question in a patient whose emotional instability is quite obvious. To operate upon such a patient will inflict real and perhaps irremediable psychic trauma to that patient. To overlook an organic lesion will condemn that patient to a life of continued suffering if not worse. Every surgeon of experience can recollect innumerable instances when he has been faced with this question. At present we have no answer. The experience and acumen of the surgeon, the maturity of his reasoning must be called into play. If he errs, he will have a sick and dissatisfied patient and he may even be the subject of implied ridicule of his fellows.

Every surgeon is faced with this problem. So we must be tolerant when we know that others are faced with similar problems. When we see patients who have the same symptoms after a cholecystectomy as before, we realize that one of our fellow surgeons faced this problem and decided it according to his best judgment. Not alone that, but now we must again study this patient from every angle to make sure again that there is not some organic disease before we label them as psychoneurotic.

The diagnosis of a psychosomatic disorder can be made only after all of the studies that can be marshalled to aid in diagnosis have been exhausted. And then we must be especially alert, lest we ascribe any real syndrome, that they may subsequently develop to their psychoneuroses.

William C. Beck, M.D.

THE SHIFTING ECONOMIC SITUATION AS A DETERRANT TO THE EDUCATION OF THE SURGEON*

ALL those interested in the future education of the American surgeon must study and evaluate the changes which modern economic situations have made in hospital practice. Twenty years ago a great many of our largest hospitals had a major number of so-called charity or free beds frequented by the poorer class where such people who could not afford the services of a private surgeon went to have their surgery performed. The teachers of surgery in the great schools were very jealous of their prerogatives as regards these "open" or charity beds because this was the material which they could utilize as they saw best in the final education of the young surgeon.

The education of the surgeon and the physician differ essentially, in that although many may profit from the examination of a patient with a cardiac murmur or some unusual medical sign, only the person who himself conducts the operative procedure can fully benefit from that experience. This handicraft aspect of surgery is something that must be learned and it must be learned under the most careful tutelage. After several years as an intern or assistant resident surgeon or fellow, the young surgeon reaches the point where he cannot progress further unless he has individual surgical responsibility and the opportunity to operate independently and by himself. The experience acquired by such operations gives to the young surgeon confidence and practice which are essential to his final development.

Shifting economic trends now are jeopardizing the existence of so-called charity or "open" beds in many of our greatest hospitals, either because the people are in large part becoming insured through group insurance systems, or because industry is taking over the care of its laborers and has its own doctors to look after them. Such patients are now private patients in the sense that a contract exists which places them under the care of specific individuals and their surgical care cannot be turned over to other than those mentioned in the contract. The number of "open" or charity beds is further diminished by the rising income of all workers which has now reached the level at which most hospitals could fill a greater percentage of their beds with paying patients. Hospital trustees, impoverished by the rising cost of hospitalization, have noted this change in the economical status of their patients and are anxious to close as many of their public beds as possible and thus cut down their mounting deficits. There can be no question but that the shift toward insurance schemes, whether public or private, is sure to continue and to advance. Whether or not it can survive a depression will depend in great part on the amount and vigor of Government aid.

Irrespective of future alterations in the economic situation, the present status must be studied and those interested in a source of material to fully train the surgeon must modify their plans for as teachers it is their responsibility to see that this important matter continues. It would appear that hospital staffs, including the teaching staffs of university hospitals, in order to cover this might organize themselves into some kind of "group practice," including in their group the senior members of the house staff so that when the hospital in which they work takes on people who are insured, whether by contract with a labor group, a bank or a business organization, it would be equally satisfactory to the insured if the resident surgeon, as the right hand

* This article was received shortly before Dr. Cutler's death and was probably one of the last editorials he wrote for publication.

man of the chief of staff, be also in the group allowed to perform the final surgical ordeal. Some such organization must be found shortly for the economic situation is affecting not only this vitally important matter of the training of the surgeon, but the education of medical students as well. The sooner the teaching profession wrestles with this problem the sooner it will be accomplished. I have no doubt that those people desiring to be insured will accept any agreements which the leading teachers wish to bring forward.

This is written only to stimulate those responsible to make such studies now and to plan carefully their educational program in the future, basing it on the utilization of insured or semiprivate cases rather than on the continuously disappearing supply of so-called charity or "open" beds.

Elliott C. Cutler, M.D.

Students who watch operations have never really been able to see or learn everything which goes on during the operation because they are usually too far away from the surgeon. To overcome this Trimble and Reese introduced television in an operating room at the Johns Hopkins Hospital, with excellent results. Even the most minute surgical procedure can now be made plain to the observer, surely a worthy advance in the teaching of operative surgery. The day is not far removed when all-important teaching centers will have at least one of their operating rooms, and its associated amphitheater, so equipped. (*Richard A. Leonardo*, M. D.)

Original Articles

POSTERIOR SPHINCTEROTOMY

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AN incision of the subcutaneous portion of the external sphincter is correctly called a sphincterotomy. Such a procedure was first advocated by Boyer back in 1788[1] for the cure of fissure in ano. It is not definitely known whether or not he recommended division of the sphincter through the base of the fissure but Earle in 1911[1] did recommend it on the theory that it was the spasm that caused the fissure and deep incision would obliterate this spasm. As approximately 90 per cent of fissures occur at the posterior commissure his incision must have been made in the latter location most of the time. Since then, other operators have considered it more logical to make the incision in one of the lateral quadrants rather than either in the base of the fissure or at the posterior commissure.

An incision of the pecten band, on the other hand, is correctly called a pectenotomy. It was first advocated by Miles[2] in 1918 who used it not only for the cure of fissure in ano but also as an integral step in his hemorrhoidectomy operation. He divided this band in the right posterior quadrant.

Gradually in this country a group of proctologists have readopted the custom of making sphincterotomy in the posterior midline[3–5,22] and their operation, often of necessity as well as by intent, has also included incision of the pecten band whenever present. In other words, they perform simultaneously both a sphincterotomy and a pectenotomy located at the posterior commissure. The reasons will become clear later on in the text.

Confusion as to the difference between sphincterotomy and pectenotomy still exists. However, this confusion disappears whenever a clearcut conception of the normal anal musculature is contrasted with the pathologic condition inherent in pectenosis. Both of these subjects can stand repeated exposition.

ANATOMY OF THE ANAL MUSCULATURE

It is only within the last few years that accurate information concerning the normal anal musculature was established by Milligan and Morgan,[6] Blaisdel,[3] Levy[7] and Gorsch.[8] Certain salient features of this mechanism should be emphasized as follows:

1. The external sphincter is composed of three divisions with the two deeper portions (i.e., the superficialis and the profundus) surrounding the internal sphincters and, contrarily, the subcutaneous portion being distal or caudal to and in the same vertical plane as the internal sphincter. (Fig. 1.)

2. The presence of and the function of the conjoined longitudinal muscle should not be overlooked. This muscle is a continuation of and a confluence of the longitudinal bands on the outside of the large intestine plus some of the fibers of the levator ani. It surrounds the internal sphincter, encases the bundles of the external sphincter and becomes inserted in part into the skin of the anus at Hilton's white line in the intermuscular

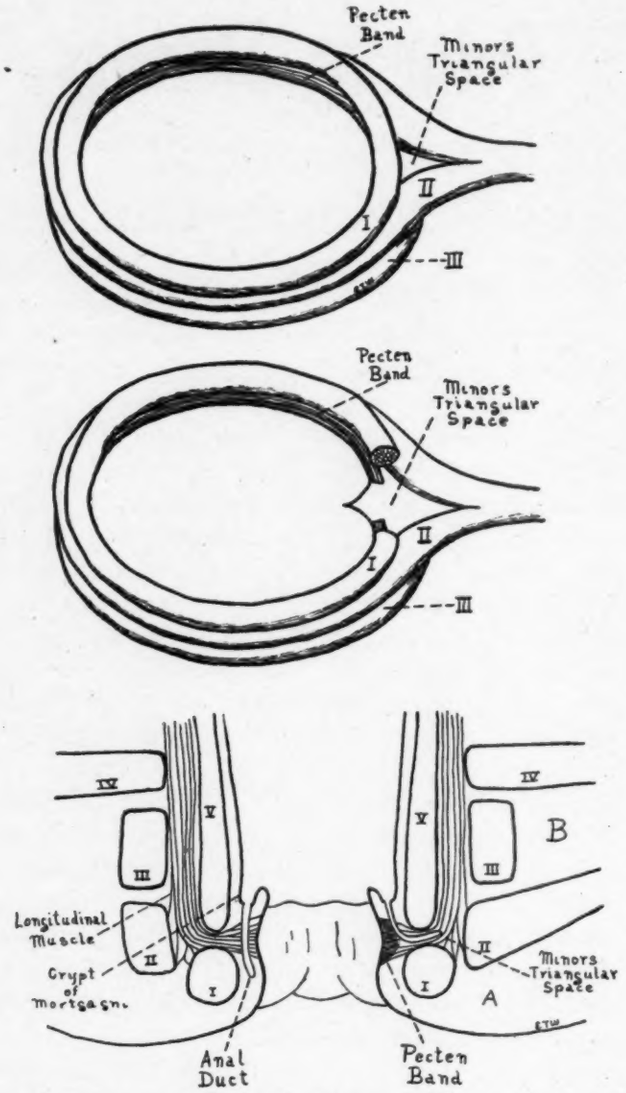

FIG. 1. Diagram illustrating the anatomy of the anal sphincters. The upper and middle plates depict the three portions of the external sphincter looking from without inward through the anus. The lower plate depicts a sagittal section through the anus and sphincters. In all three plates I represents the subcutaneous portion of the external sphincter; II represents the superficial or superficialis portion; III is the deep or profundus portion; IV is the levator ani muscle while V is the internal sphincter. The upper plate shows the doughnut-like character of the subcutaneous and deep portions of the external sphincter and the way the tangential legs of the superficial portion go off to produce the anococcygeal attachment with Minor's triangular space between the two legs. The middle plate shows how the latter space is opened up by a posterior sphincterotomy. The lower plate shows the "directive" influence of the longitudinal muscle on the anal glands both laterally (left) and at the posterior commissure (right). It should be noted that there is a distinct difference between an anal duct (called a "crypt" when infected) and the crypt of Morgagni. In this plate the anal duct or "crypt" is pictured opening into the crypt of Morgagni as it does in the human anus. Spaces A and B are the superficial and deep post-anal spaces, respectively, with which Minor's triangular space communicates. The location and strangulating influence of a pecten band is clearly seen.

septum.[4–6,8,22] (Fig. 1.) This muscle becomes more fibromuscular in character the nearer it approaches the skin of the anus and the latter characteristic is an important factor in the production of some of the pathologic conditions to be described. As Gorsch says, "these longitudinal fibers are of special importance on account of their directive influence on the extension of infectious processes from the pecten and the rectum to the perianal tissues."[8]

3. There are several perianal spaces which, although they are very important in the surgery of this region, have seldom been given the notice they deserve. The most important one is the actual or potential space called Minor's[9] triangular space lying between the two tangential legs of the superficial portion of the external sphincter as the latter go off to become attached to the coccyx. (Fig. 1.) For purposes of simplification it may be stated that the deep and subcutaneous portions of the external sphincter form doughnut-like rings above and below this space, respectively. Technically, the latter should be called the posterior intermural space to differentiate it from the superficial and deep postanal spaces which are below and above the tangential legs of the superficial external sphincter,[8] respectively, and with which it potentially communicates. (Fig. 1.) This potential communication is posterior to and outside the two doughnut-like deep and subcutaneous portions of the external sphincter. In addition to these three spaces there are of course the two ischiorectal spaces on each side of the anus and rectum bounded above by the levator ani muscles, by the skin below and by the pelvis and rectum on either side.

PECTENOSIS

A pecten band (pectenosis), on the other hand, is a pathologic structure. It was first mentioned and given its name by Miles[2] in 1918 but was studied and described in greater detail by Abel[10] in 1932. It pertains to a fibrotic band occurring in and beneath the transitional skin of the anus in the region of the intermuscular septum. The etiology of this condition had been variously laid at the door of both chronic passive congestion and chronic infection. However, when the classical work of Tucker and Hellwig[11] confirmed by Hill, Shryock and Rebell[20] put onto a firm foundation the presence of some infected anal ducts ("crypts") beneath the skin of the anus, it was possible to incriminate these latter structures as being the prime causative agents. Certainly they can be demonstrated to exist coincidentally with a pecten band in practically all cases whenever the latter is found to be present. Apparently the fibromuscular insertions of the longitudinal muscle are transformed into fibrous insertions in the presence of and as a result of chronic infection existing within and around these anal ducts,[5] and gradually the intermuscular septum becomes filled with and replaced by a band of pearly white connective tissue.[22]

Contraction of this fibrosis produces several pathologic effects. First, it strangulates the neck of the anal ducts as the latter pass between the fibrotic fibers, turning the ducts into little undrained abscesses which have a tendency to burrow and thereby produce most of the various perianal and perirectal abscesses, blind fistulas, complete fistulas and fissures. Second, in due course of time this dense contracted band of fibrosis within the anus prevents proper relaxation of the sphincters. This can proceed to such an extent in elderly patients that the anus actually becomes a stricture. Third, it either anchors the skin inside the anus onto itself or turns it into a fibrous type of tissue which is unelastic, avascular, splits easily and gives rise to constant discomfort.[12,22]

Therefore, the finding of fissures, abscesses, posterior fistulas, pecten bands and even chronically sore "behinds" usually infers the presence of some acute or chronically infected crypts.[3,5,11,12,15–20,29–31]

It might be argued that whenever a pecten band is encountered, simple pecte-

notomy would be all that was necessary to sever it and lay open to drainage the underlying infection but this does not take into account the anatomy of the sphincter at the posterior commissure. As we have shown[5] infected anal ducts in the latter location have a tendency to be "directed" by the longitudinal muscle into Minor's triangular space and it requires not only a pectenotomy but also a posterior sphincterotomy to adequately drain this space.[9]

INDICATIONS FOR POSTERIOR SPHINCTEROTOMY

Inference as to the indications for sphincterotomy located at the posterior commissure has been made in the preceding sections but for those not versed in proctology a more detailed exposition will follow. We personally have performed such a procedure in over 800 patients either in connection with the several conditions mentioned in the previous and following sections or as we found that it had secondary advantages when done in addition to some proctologic operation. Our experience has shown it to be indicated in the following conditions: (1) cryptitis; (2) strictures about the anus (3) posterior perianal abscesses; (4) complete fistulas at the posterior commissure; (5) blind fistulas at the posterior commissure; (6) posterior fissure in ano; (7) hemorrhoidectomies; (8) certain cases of pruritus ani; (9) for operative intrarectal exposure.

Prior to taking up these items in more detail we wish to emphasize that whenever the term posterior sphincterotomy is used henceforth in this article, it will be assumed that pectenotomy is included if pectenosis is present.

Cryptitis. When it is more generally realized how important these infected anal ducts or crypts are in the production of so much anal and perianal pathologic conditions, a search for their presence will be more frequently made and the cryptectomy operation will become a more common procedure. Our conception of a crypt is simply that of an anal duct which was originally of microscopic size but which became enlarged and elongated due to repeated bouts of infection. During the course of this process and consequent thereto pectenosis or a pecten band was produced which partially or completely strangulated the neck of the crypt thereby obliterating adequate drainage and giving rise to further elongation and enlargement as well as symptoms.

The term crypt is used very loosely and is surrounded by much confusion in the literature. This is because there are two crypts—the crypts of Morgagni and the aforementioned infected anal ducts commonly called crypts. The crypts of Morgagni are simply the cup-like depressions to be found behind the mucocutaneous or pectinate line in the bases of the sulci between the columns of Morgagni. They are wide open, shallow depressions of varying but minor depth and it is hard to conceive how they could burrow their way down behind the pecten band which is distinctly below or distal to them. The anal ducts on the contrary have their openings in the crypts of Morgagni but extend therefrom downward through the fibers of the longitudinal muscle or pecten and into or among the various members of the anal sphincter. In the lower animals they are present in greater numbers and exist as definite secretory glands. After they become infected they are generally spoken of as crypts. This terminology has come about through popular usage and arises from the fact that the terms crypt and cryptitis are shorter and more easily enunciated than anal duct and anal ductitis and because they were formerly thought to be the crypts of Morgagni. If this difference is kept in mind, all confusion disappears. To repeat, the cup-like depressions between the columns of Morgagni are the crypts of Morgagni while a crypt on the other hand is an *infected* anal duct which empties into the crypt of Morgagni. As previously stated this conception is based on the pathologic studies made by Tucker and

Hellwig,[11] Hill S. Shryock and ReBell[20] and others.[5]

The cardinal symptoms of cryptitis are burning, irritation or aching plus periodic attacks of anal soreness. Crypts can only be demonstrated by means of a crypt hook. Therefore, let those critics who do not own such a hook or who do not use it make no doubtful or disparaging remarks concerning the presence of these pathologic structures especially in the light of the vast amount of literature concerning them which has appeared in the past few years.[3, 5, 11, 12, 15–22, 25–31] Furthermore, although the recognition of their importance in the production of proctologic pathologic conditions may be a moderately recent development, nevertheless their presence in the adult human anus was known and recorded back in 1732 by Winslow.[23]

Generally whenever one crypt is found, there are usually at least two more and always one of these is at the posterior commissure and more often than not the latter one has its distal tip pointing into Minor's triangular space.[5] (Fig. 1.) We used to excise the one offending crypt in the clinic or office in an ambulatory manner[19] but so often we found that while we may have eradicated one of the crypts a second one and even a third one was lighted up by the procedure, consequently more operations became necessary much to the patient's annoyance. Furthermore, a crypt at the posterior commissure with its tip out in Minor's triangular space was not always eradicated by such a simple procedure. Consequently, we now put these people into the hospital and do a thorough, multiple cryptectomy plus posterior sphincterotomy.

Furthermore, not too rarely a patient will be seen who gives a history wholly consistent with that of fissure in ano but no fissure can be found. However, at the posterior commissure is found an area of exquisite tenderness, induration, a pecten band and even spasm of the sphincter. The crypt hook can be pushed down through a cryptic opening, beneath the pecten band and into the area of induration. Excision of all of the tissues on the hook plus posterior sphincterotomy will affect a cure. Waiting for nature to take its own course leads to either a fissure or a fistula. Also, not too rarely, the proctologist meets a patient who has had a hemorrhoidectomy unsuccessfully performed for the relief of symptoms consistent with cryptitis. Examination reveals the offending crypt still at the posterior commissure but now also ensnared by the fibrosis produced by the hemorrhoidectomy. Cryptectomy plus a posterior sphincterotomy gives permanent relief.

Strictures about the Anus. As we and others have pointed out many a case of long-standing, contracted anus is the result of this chronic infection of the anal ducts or cryptitis. However, other causes for anal and lower rectal stricture occur such as past or recurrent abscess formation, chronic fistulas, false appositions following hemorrhoidectomies, lymphogranuloma venereum, congenital malformations, etc. Whatever the cause for a stricture in this location, it will be found that a posterior sphincterotomy not only increases the size of the anus but lays it open long enough to allow for resolution of a great deal of the fibrosis and to give time for epithelialization to take place over the ends of the severed stricture thereby preventing premature reapposition. When the stricture is somewhat above the anus, as in chronic abscess formation for instance, it can be incised as a part of a posterior sphincterotomy and then after control of bleeding the superior edge of the mucous membrane can be brought down and anchored into the base of the wound at the level of the internal sphincter or erstwhile pectinate line.[24]

Posterior Perianal Abscesses. Perianal abscesses near the posterior commissure practically always have an internal opening in a crypt.[15–18, 22, 25, 27, 29, 30, 31, 33] The latter is usually at the posterior commissure. Its opening became strangulated by the fibrosis of the longitudinal muscle fibers[5] and

the infected stricture burrowed its way either down between the mucous membrane and the external sphincter or out through Minor's triangular space into the superficial postanal space and thence into the perianal tissues. (Fig. 2, middle plate.) considered best by most operators to simply incise the abscess and await the subsequent production of the fistula. Incision or excision of such a fistula will usually require total incision of the subcutaneous portion of the external sphincter at the

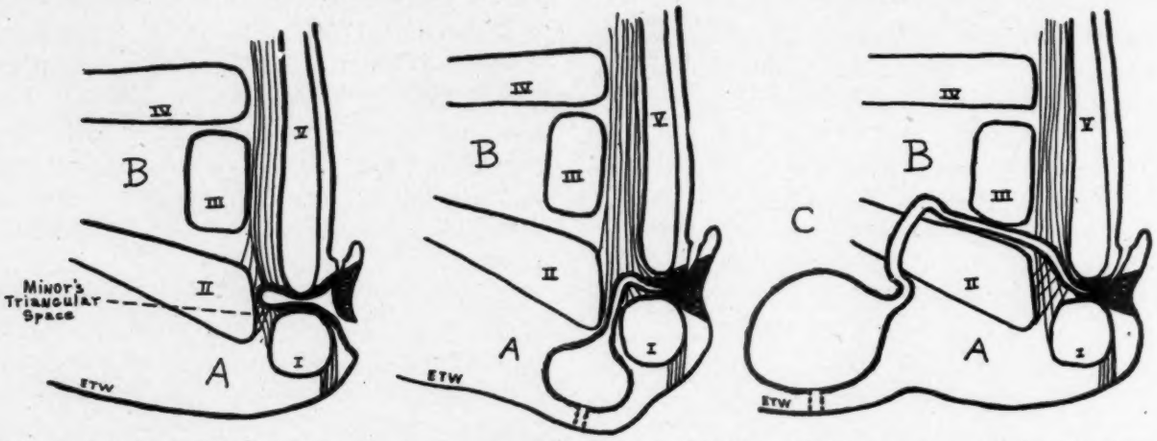

Fig. 2. Diagrams illustrating the varying progress of a strangulated crypt. All plates represent sagittal sections through the posterior commisure. The left one depicts the production of a fissure-in-ano (so-called). Really it is an intra-anal fistula; a crypt became infected, produced a pecten band, was strangulated by the pecten band, burrowed back toward or into Minor's triangular space and finally ruptured out into the anus lower down. The middle illustration is that of a post-anal abscess and fistula with the crypt making its exit from Minor's triangular space upward into the deep post-anal space and thence to the right or left into the ischiorectal space.

Following incision of the abscess, a crypt hook can often be introduced into the internal cryptic opening and forced down through the pecten band and on into the abscess cavity. Incision on the hook is made and is followed by a full posterior sphincterotomy taking off as much skin from the other buttock as has been laid open on the affected side. (Fig. 3.) A permanent cure is had and a secondary operation for a fistula is eliminated. No deformity and hardly any scar will follow.

Complete Fistulas at the Posterior Commissure. The vast majority of ischiorectal abscesses have an internal opening in a crypt.[33] When this crypt is at the posterior commissure, it has a tract running down behind the pecten band, backward through Minor's triangular space and then either upward into the deep postanal space and thence into the ischiorectal space or downward into the superficial postanal space and thence into the latter space. (Fig. 2.) Such tortuosity is too much for any unyielding probe; consequently, it is posterior commissure. This should include the severing of all of the pectinotic fibers in order to secure a smooth, undeformed and elastic anus afterward.

Submucous fistulas and abscesses in this location should also have the benefit of more adequate drainage and obliteration by means of sphincterotomy and pectenotomy after the incision of the tract higher up has been completed.

Blind Fistulas. Not too uncommonly a blind fistula is found at the posterior commissure. Its one opening is usually at the level of the pecten band and allows only partial drainage either into the anus or into the rectum or both. The cavity behind the pecten band may be small or large but the production of seropurulent discharge is often voluminous. For both exploration and cure a right angled probe is necessary. (Fig. 3.) Here again it should be stated that following its eradication the remaining fibers of the pecten band should be incised in order to obtain a smooth, elastic anus once more.

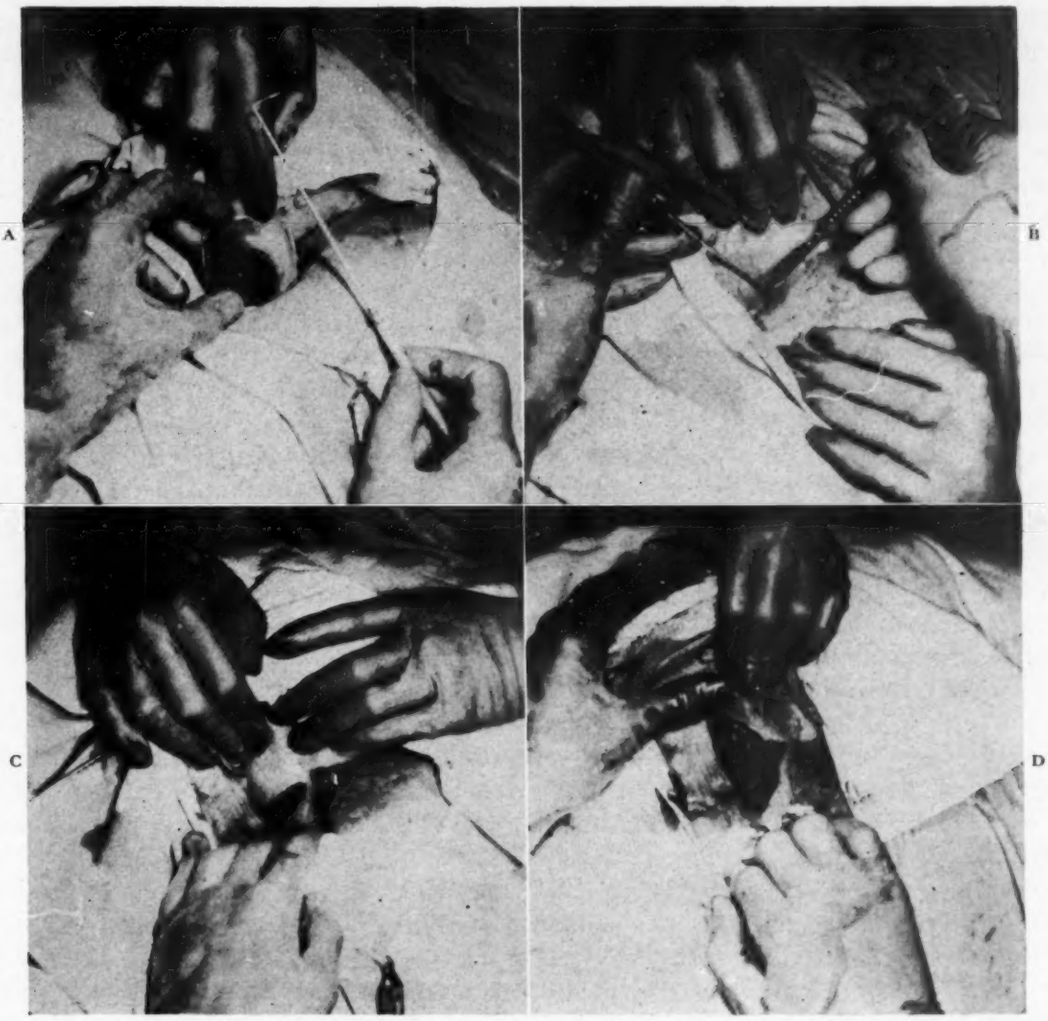

Fig. 3. Illustrating the cure of a blind fistula at the posterior commissure. A, a properly made right angled probe with a speculum inserted into the anus; B, the hook has been introduced into the fistula, the speculum has been withdrawn and the scalpel is incising down onto the hook; C, the wall of the fistula has been excised and a posterior sphincterotomy plus a pectenotomy have been completed. D, shows how much additional skin must be excised from either side in order to delay healing until complete granulation has taken place. The resulting scar will be in the base of the commissure and will be hard to find within three months.

Fissure in Ano. In the opinion of several American proctologists sphincterotomy, or at least part of it (pectenotomy), is a "must" in fissure work.[3–5,9,10,12,22] Both theoretically and practically it fills the need for a procedure which will definitely cure and as well definitely prevent the frequent recurrences which so often follow less radical procedures, such as simple excision, dilatations, cauterization, injections, etc. Sometimes the use of a right angled probe shows the fissure to be a true blind fistula going back into Minor's triangular space.[5] Such a finding confirms the reasoning that a fissure is simply an infected crypt which has ruptured through the anal wall. (Fig. 2.) The fact that 95 per cent of fissures occur at the posterior commissure means something and this something has to do with cryptitis plus the difference in the anatomy of the sphincters at the posterior commissure. As previously described crypts in the latter location have their ducts directed backward by the longitudinal muscle toward and into Minor's triangular space and a pecten band turns

any infection in the duct into an intermural abscess. (Fig. 2, left plate.) During infection this little abscess is compressed and extension of its contents is brought about. This extension follows the line of least resistance and in fissure cases this line appears to be through the thin intra-anal skin just below the pecten band. Therefore, a fissure usually should be considered and treated as being a fistula with its primary opening in the original mouth of the crypt up in the rectum and its secondary opening below the intermuscular septum. The tract meanwhile traverses backward behind the pecten band toward if not into Minor's triangular space. (Fig. 2.) Cure requires excision of the whole tract, plus drainage of Minor's triangular space plus time enough for granulation to take place before full re-epithelialization occurs. Posterior sphincterotomy accomplishes all of this.

Hemorrhoidectomies. There is one school of thought headed by Miles and Abel which considers pectenosis to be the result of chronic passive congestion accompanying or following hemorrhoids. Careful and prolonged observations of over a thousand cases of hemorrhoids has convinced us that the majority of hemorrhoids are not accompanied by pectenosis except when there is also a coincidental cryptitis. However the case may be, some proctologists advocate routine posterior sphincterotomy in hemorrhoidectomies. We go further. We believe it to be a must in this operation[14] for several reasons: (1) It tends to prevent false re-appositions and thus stricture formation; (2) it definitely diminishes postoperative edema plus the pain and discomfort that accompanies the latter; (3) it abolishes spasm of the external sphincter allowing earlier, freer and more comfortable bowel movement; (4) postoperative skin tabs are less likely to form; (5) a simple pressure dressing applied into the resulting wide open anus prevents any oozing from those tissues which are distal to the transfixed hemorrhoidal pedicles.

Elimination of postoperative edema alone is an adequate indication for its routine use in hemorrhoidectomies for this edema is probably the greatest factor in the production of disability, prolonged hospitalization and morbidity following hemorrhoidectomy operation.

Pruritus Ani.[10] Spiesman,[13] J. W. Morgan[12] and others have advocated pectenotomy for pruritus ani. Gorsch finds posterior sphincterotomy theoretically indicated in cases accompanied by cryptitis but does not wax enthusiastic about it. We have not had much success with it in pruritus ani when performed on an empirical basis. However, when a case is encountered which has undergone irreversible changes in the form of large folds of hyertrophied skin alternating with deep creases, no relief and certainly no cure can be had until the skin is smoothed out. Excision of the folds, undercutting of the remaining skin strips and deep, wide posterior sphincterotomy followed by frequent digital prevention of too rapid healing results in a smooth, re-epithelialized perianus which has the form of an inverted funnel. Treatment for the pruritus then has some chance of success.

Operative Intrarectal Exposure. Many surgeons, especially non-proctologists, routinely dilate the sphincters to get more room. Occasionally they have an uncomfortable period of three to six months while waiting the return of continence in some one of their patients. We believe that much more adequate exposure without the possibility of such a complication can be obtained by means of sphincterotomy. However, when as much intrarectal exposure as possible is desired, the incision should be made in either the right or left latero-anterior quadrants as this gives more room than does a posterior sphincterotomy.

SURGICAL PROCEDURE

Incision of the muscle is easily done with the patient in any of the usual rectal operating positions—Simms, prone or litho-

tomy. The tip of the left index finger is pressed into the intermuscular septum or above the pecten band if present and the subcutaneous external sphincter is palpated between it and the thumb. Then, while both buttocks are held apart and the skin of the posterior commissure is put on tension by an assistant, incision is made down the side of the finger, through the skin, through the "bar" of subcutaneous external sphincter, through the pecten band and back into Minor's triangular space lying between the two tangential legs of the superficial external sphincter. Some operators, however, like to insert a Kelly forceps between this portion of muscle and the other sphincter bundles and incise down onto the forceps.

Experience soon reveals when the internal sphincter has been reached and when all of the pecten band has been separated. Complete and adequate incision is had when the base of the wound is composed of soft, elastic muscle. No horizontal strands of fibrous tissue should be left. Finally the skin edges on each side are cut well back, (Fig. 3) and bleeding is carefully controlled. There is usually at least one active bleeder and experience has caused us to emphasize its *transfixation* as an ordinary tie has a tendency to slip off of the fibromuscular tissues. All raw areas are covered by gauze impregnated with sulfathiazol ointment and pressure is applied by means of adhesive (not a T-binder). The dressing is changed in twenty-four hours.

The anesthesia varies with the condition encountered. Most fistulas and most abscesses call for a spinal while hemorrhoids, crypts, fissures and some strictures can be done, if desired, under regional novocain infiltration accompanied by oil anesthesia in the sphincters and out in the buttocks. The latter allows the passage of a postoperative stool sooner and with less discomfort. Incidentally, we find the Angelo[32] method of obtaining an early postoperative stool without aid of oil or enema to be quite successful. He advocated the administration of sulfathaladine pre- and postoperatively with encouragement of a spontaneous evacuation on the second or third day. The ensuing stool is soft and mushy but gives a decided signal withal.

Results are uniformly satisfactory. Healing takes place in from two weeks to two months depending on the patient, the pathologic condition and the width and depth of the incision. Often after a few months not even a scar can be seen as the posterior commissure is the site of embryonic fusion of two skin surfaces and reepithelialization or refusion seems to take place without scar formation just as well in adult life as in embryonic life. Sometimes the whole anus becomes quite unelastic or "splinted" for three or four months but softening and relaxation eventually takes place. We have had only two patients who expelled gas involuntarily for a time. The end result in even these two patients was as satisfactory as in all the others. One clinic patient had to be given large doses of cevitamic acid before the sphincters would heal together again. Care must be exercised in the use of mineral oil as more than ½ ounce daily may leak or produce involuntary stools before the sphincters have reapposed. Several stools following posterior sphincterotomy for fissure in ano are not unusual due to the release of spasm, pain and fear. On the whole, we personally are so enthusiastic about this procedure that we would be at a loss to know what to do adequately without it in many situations in which we now use it as a routine measure.

CONTRAINDICATIONS

There are two conditions in which posterior sphincterotomy is contraindicated; one is functional in character and the other, anatomic. The first one is the ataxic sphincter. It is seen only rarely but not so rarely but what one of our colleagues believes that he did a posterior sphincterotomy in the presence of such a patulous

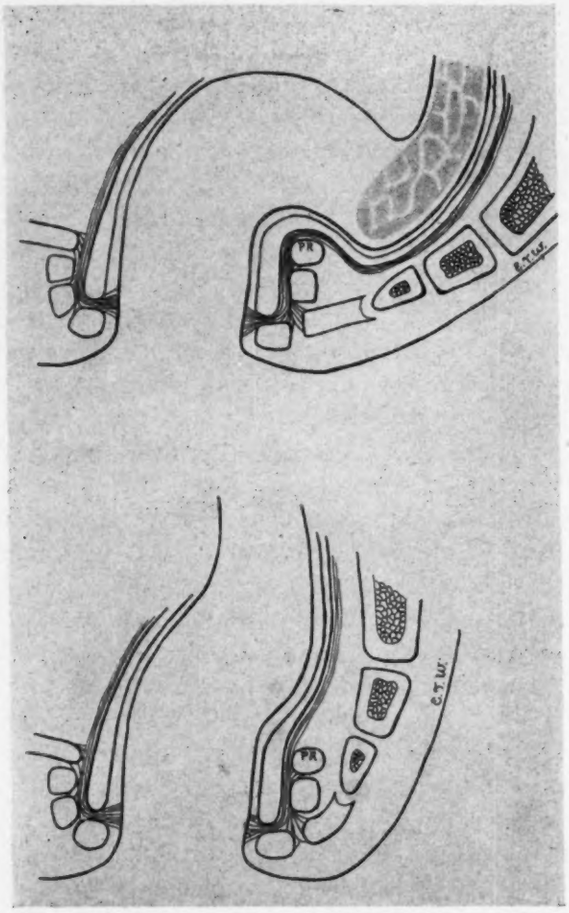

FIG. 4. Illustrating two extreme examples of a recto-anal angle. The top plate shows the constipative type. There is an acute angle at the recto-anal junction caused by a markedly concave sacrum carrying the rectal ampulla well backward while a tense puborectalis portion of the levator ani muscle pulls the anus well forward. (PR. in the illustration indicates the puborectalis, a sling-like portion of the levator ani muscle). Expulsive forces have a tendency to exert their action behind the anus rather than down through the anus. Consequently, such an acute angle places an added difficulty on the progress of a hard stool. The lower plate shows the prolapsive type. There is no angle due to a more or less vertical sacrum and a lax puborectalis sling. Prolapse of the mucous membrane or even procidentia is a frequent complication.

anus, with some incontinence as a result for an unreasonably long time. Therefore, whenever the anal musculature fails to close quickly after digital examination or whenever it is found on digital examination that the usual tonic pressure of the sphincters on the finger is lacking, the test for an ataxic sphincter should be made. This is done by forcibly inserting three fingers into the anus and deliberately separating them thereby dilating the sphincter without aid of anesthesia. If the patient accepts this insult without complaint nor attempts the escape of the fingers, then the latter are quickly removed and the anus will remain widely open especially if a little traction is made on the buttocks. The popular conception concerning this abnormal functioning of the anus puts it into the class of syphilis, but we have exhaustively examined several such cases and agree with other authors that no known etiology has as yet been found which adequately explains the findings.

The second condition is anatomic. It is the absence of an anorectal angle. (Fig. 4.) Normally the puborectalis portion of the levator ani muscle forms a sling around and behind the upper anus and lower rectum pulling them forward and thereby producing a definite angle posteriorly. The examining finger at first passes up the anus in a direction pointing toward the navel but, whenever there is a definite anorectal angle, the finger subsequently must be bent posteriorly and point toward the concavity of the sacrum due to the changed direction of the lower rectum. In extreme cases the angle is markedly acute and a definite pouch exists behind the anus anteriorly to the coccyx and sacrum. However, occasionally the other extreme is encountered wherein there is no angle and the finger continues to go straight up. (Fig. 4.) It is in such cases that prolapse and procedentia are prone to occur as this anorectal angle or shelf has a markedly rstraining influence on any tendency for the mucous membrane or rectal wall to intussuscept through the anus. In the absence of this restraining influence, nature has difficulty in preventing extrusion of the rectal wall whenever she attempts to expel rectal contents. A posterior spincterotomy would only add to nature's difficulties.

SUMMARY

The difference between posteriorsphincterotomy and pectenotomy is described,

together the accompanying anatomy and pathologic conditions, respectively.

Indications for and contraindications against posterior sphincterotomy are discussed in detail.

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CLINICAL STUDIES OF LIVER FUNCTION

THE HEPATORENAL SYNDROME

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In a previous study the effect of anesthetics and a variety of elective surgical procedures on liver function in eighty-four apparently healthy patients was investigated.[28] Spinal anesthesia, in the absence of a fall in blood pressure, and local anesthesia (0.5 per cent novocain) together with amytal and morphine produced no detectable alteration in the functional state of the liver following surgery. Avertin given as a basal analgesic in combination with novocain infiltration reduced liver function by 5 to 30 per cent. Open drop ether anesthesia produced transient subnormal hepatic function in all patients studied.

The present study was undertaken with the view of evaluating certain pre- and postoperative therapeutic measures directed at improving the functional state of the liver in critically ill, poor-risk, surgical patients. Because of the high incidence of associated renal and hepatic insufficiency (hepatorenal syndrome) following operation, an evaluation of the rôle of the liver in postoperative morbidity and mortality must of necessity include serial studies of both renal and hepatic function.

LIVER DEATH AND THE HEPATORENAL SYNDROME

A comprehensive review of the growing literature on the subject "liver death" and the "hepatorenal syndrome" is beyond the scope of this paper, particularly in view of the exhaustive review by Wilensky[35] and Boyce's excellent monograph on the rôle of the liver in surgery.[5] The present study, begun in 1940 and interrupted by World War II, confirms in somewhat greater detail and extends in some instances certain of the latter author's clinical observations.

An intimate relationship between the liver and kidneys has been recognized for nearly a century; however, Heyd's description in 1924 of liver death following operations on the biliary tract appears to be the first clear-cut account to justify the concept of "liver-kidney syndrome."[11] In a later analysis of mortality following operations on the biliary tract Heyd[12,13] classified his clinical material into three groups:

Type 1. In this group the patients are considered good operative risks, are not jaundiced and are subjected to simple cholecystectomy. Almost immediately from the time of operation there is a continuously ascending temperature, a rapidly developing lethargy, followed by stupor and coma with death terminating the picture in from eighteen to thirty-six hours.

Type 2. These patients are operated upon for relief of obstructive jaundice. Convalescence is normal for four or five days when, in spite of diminishing jaundice, the patient becomes delirious, then stuporous and finally comatose. Death occurs not unlike the cholemic death that occurs in unrelieved obstructive jaundice.

Type 3. These patients are not jaundiced, have what is considered normal kidney function and are considered good risks. Forty-eight hours after operation the pulse rate becomes elevated and the patient quite rapidly presents a picture of shock followed by decreased urinary output, then anuria with uremia, fever, coma and finally death.

A more detailed classification of the hepatorenal syndrome under essentially the same general headings as Heyd's has been suggested by Boyce.[5]

Boyce and others have pointed out that postoperative deaths of the above types are

in no way restricted to patients operated upon for biliary tract disease. A number of reports may be found in the literature in which the hepatorenal syndrome occurred after liver trauma[14,15,17,30] following ligation of a hepatic artery,[31] after hysterectomy,[8] after colostomy,[8] after resection of the cecum,[5] following intestinal obstruction,[35] following severe burns[3,5,7,36] and even after interval appendectomy.[5]

While the terms "liver death" and "hepatorenal syndrome" have been accepted by the majority as clinical entities, the exact nature of the breakdown of liver and kidney function remains obscure. Death follows removal of the kidneys from experimental animals due to accumulation of toxic nitrogenous excretory products. But, as yet, no investigator has been able to demonstrate the cause of death in animals with livers removed. Postmortem studies of liver and kidneys from patients whose fatal clinical course conforms with the classical picture of hepatorenal syndrome frequently reveal lesions, degenerative in character which seem inadequate to account for the fatal outcome. Touroff[32] warns against the clinical diagnosis of "liver death" in patients which develop unexplained coma and rapidly mounting fever after biliary surgery unless adequate autopsy excludes other causes. In reviewing 1,360 cholecystectomies, Touroff found only four cases which, following operation, developed without apparent cause the classical syndrome of rapidly rising temperature and shock with early fatal termination. A clinical diagnosis of "liver shock" seemed justified in all four; however, autopsy revealed an overwhelming infection to be the cause of death in all instances. Two had subphrenic abscess, one a gangrenous bronchitis with bilateral pneumonia and the other a severe hemorrhagic pneumonia.

Boyce[5] is of the opinion that hyperpyrexic liver death and liver-kidney syndrome are a single pathologic entity, the difference in the clinical course being determined by the magnitude of hepatic distintegration. Failure of hepatic detoxication throws a strain on the kidneys which are in turn overcome by toxic products ordinarily rendered innocuous by a normal liver. According to this author, hepatic changes are always followed by renal lesions if the patient lives long enough.

Helwig and Orr[16] have studied in detail the blood chemistry in patients with severe traumatic liver injuries and presenting the clinical picture of the hepatorenal syndrome. Their interpretation of the causative basis for kidney failure secondary to autolysis of liver tissue differs from Boyce's only in that they ascribe the necrotic renal changes to a specific toxin of liver origin. Boyce's experimental production of degenerative changes in the liver and kidneys of dogs by the injection of saline extracts of liver from a patient who died a liver death thirty-six hours after cholecystectomy would seem in favor of a specific toxin. Control experiments in which extracts of normal liver were injected were without effect on liver or kidneys.[5]

PRESENT STUDY

In the hepatorenal syndrome two organs are involved—the liver and the kidneys. In reviewing the literature on this subject we have been impressed by the paucity of data pertaining to tests of both hepatic and renal function *before* operation or the onset of untoward symptoms. Conclusions are based in numerous instances on autopsy material, substantiated in many cases by determinations of icteric index and blood NPN during the fatal illness. The functional states of the two organs concerned *prior* to the onset of decompensation was unknown. Many writers infer normal renal reserve if the blood NPN is within normal range, a conclusion certainly unwarranted. Only after complete breakdown of kidney reserve does the NPN become elevated. Before the nature of the hepatic and renal insufficiency can be evaluated one must know something of the functional state of the respective organs *before* the onset of clinical symptoms. These conditions have

been met in the patients we have studied. Serial function tests were made before and after the onset of the hepatorenal picture.

TESTS OF HEPATIC AND RENAL FUNCTION

Liver Function. Five tests which measured different hepatic functions were used in the present study. These were: the hippuric acid test, the bromsulfalein dye elimination test, prothrombin estimation, and in patients with T-tube drainage of the common duct, determinations of bile salt concentration in the hepatic bile. Quantitative estimations of blood bilirubin were also made in many instances.

The hippuric acid test[21] is concerned with two hepatic functions: synthesis (glycine) and conjugation (glycine coupled with administered benzoic acid forms hippuric acid). To be valid, the hippuric acid test can be used only in patients with adequate kidney function. This limits its usefulness in studying the hepatorenal syndrome. In the presence of impaired renal excretion a subnormal hippuric acid test could be the result of both hepatic and renal insufficiency, or the latter alone. The majority of our patients were too ill to tolerate the oral test. In many continuous gastric lavage was being used. In such instances the intravenous modification of the hippuric acid test was used.[24]

The bromsulfalein (BSP) dye removal test measures the ability of the reticuloendothelial system, including the Kupfer cells in the liver, to remove injected dye from the circulation. That portion of the dye removed within the liver is transferred to the hepatic cells and is excreted in the bile. Normally a goodly portion is excreted by the kidneys. In patients with high grade *renal* retention or in the presence of a failing circulation, the results of the BSP test cannot be attributed wholly to the functional state of hepatic cells. Tests were made using the fractional modification recommended by Deutsch.[9]

Prothrombin, an enzyme or pro-enzyme essential for the normal clotting of blood, is apparently manufactured only within the hepatic parenchyma. Liver insufficiency is accompanied by decreased prothrombin production and an alteration in the clotting time of blood or plasma.[6,23,29,34] Allen's serial dilution modification of Quick's one-stage technic was used.[1]

Determination of bile salt concentration in the hepatic bile of patients with T-tube drainage offers an extremely sensitive index of functional activity in the hepatic cells. Bile salts are manufactured from protein in the liver exclusively. Minor degrees of liver injury not detectable by the usual function tests are followed by decreased synthesis of bile salts. In states of severe liver damage their synthesis may cease entirely.[4,10,19,20,26] Bile salt determinations were made using the method of Rheinhold and Wilson[27] adapted to the photolemeter.

Blood bilirubin was determined by Hoffman's photolometric modification of the van den Bergh method.[18]

Kidney Function. Two criteria for evaluating the functional state of the kidneys were used. Serial determinations of urea clearance[33] and blood determinations of non-protein and urea nitrogen were carried out at frequent intervals. In certain patients, quantitative determinations were made on twenty-four-hour urine collections for total, non-protein and urea nitrogen.

CASE REPORTS

For convenience in presentation the case reports are arranged in the following groups. The majority do not fit unequivocally into their respective group: (1) Early hepatic insufficiency followed by renal failure. (2) renal insufficiency with hepatic insufficiency and (3) associated hepatic and renal insufficiency.

1. *Early Hepatic Insufficiency Followed by Renal Failure.* Case 1. L. R., a white female, age sixty-two, was admitted on the medical service. For ten months there had been symptoms of anorexia, upper right quadrant sore-

ness, bloating, generalized weakness and a weight loss of 30 pounds. There was selective dyspepsia for greasy foods and moderate shortness of breath on exertion.

Physically the patient had a sallow, chronically ill appearance. The blood pressure was 120/80, pulse rate 96 and regular. Except for the abdominal findings and obvious weight loss the physical examination revealed nothing unusual. The abdomen was tympanitic and a soft elongated, tender mass was felt to the right of the umbilicus.

Laboratory data were as follows: On admission the hemoglobin was 75 per cent, red blood count 3,770,000, white count 14,500 with normal differential. Urinalysis revealed no unusual findings. Gastric analysis showed free HCl 39, combined acidity 43.5, total acidity 82.5. The icteric index was 10. Agglutination tests for undulant fever and typhoid were negative.

The gallbladder could not be visualized after dye. X-ray of the stomach and gastrointestinal tract revealed no abnormalities except an atonic colon. A clinical diagnosis of cholangitis was made. For thirty days the patient was treated medically. During this time the temperature spiked daily to from 100° to 102°F., frequently with an associated chilly sensation and on one occasion there was a frank chill. The upper abdominal tenderness persisted but there was no colic or severe pain. During this period of observation the icteric index fluctuated between 10 and 24.

Gallbladder drainage was advised. The patient was considered a poor surgical risk but the prognosis was considered equally poor without surgical intervention.

Further laboratory studies revealed: hippuric acid test 78 per cent, prothrombin 33 per cent, fasting blood sugar 111 mg. per cent, urea clearance 68 per cent, urine urobilinogen positive in dilution of 1:80. Although the septic temperatue continued, clinical improvement during the next eight days was observed on a regimen of high carbohydrate, low fat diet augmented by dextrose in fruit juice, bile salts (bilron), liver extract orally, vitamin B concentrate, magnesium sulfate and 1,000 cc. of 10 per cent glucose with 6.6 mg. thiamine chloride intravenously daily.

Vitamin K tablets daily for six days raised the prothrombin value from 33 to 62 per cent. (Table 1.) The preoperative hippuric acid test

TABLE 1

CASE I, L. R.

Days Pre- and Postoperative	15	4	1	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Temperature	101^{5}	102^{2}	102^{6}	97^{0}	98^{1}	100^{0}	99^{6}	98^{0}	98^{6}	98^{0}	98^{0}	97^{8}	98^{2}	98^{3}	98^{0}	98^{3}	98^{4}	98^{0}	98^{2}	98^{4}	98^{0}	98^{6}	99^{0}	99^{3}
NPN. mg. %	39	32	30	....	35	....	33	....	....	....	....	46	....	....	....	....	....	....	....	....	110	....	....	106
Hippuric acid %	78	..	82	....	....	....	70																	
Urea clearance %	68	..	70																					
Prothrombin %	33	45	58	62	54	....	54	....	....	....	....	....	....	....	....	....	....	....	....	....	....	22		
Icteric index, units	..	10	..	....	....	....	22	....	....	....	....	....	....	....	....	....	....	....	....	....	....	22		
Fluid intake, 24 hr./cc.	..		..	3000	1800	3000	2500	3200	4000	3300	2400	1200	2400	3500	2000	1800	2500	1500	1000	1700	2000	3000	3000	2000
Urine output, 24 hr./cc.	..	..	..	400	300	810	1200	520	570	500	600	300	780	160	380	620	370	340	340	440	710	660	550	465
Parental glucose, Gm.	..	..	..	200	100	200	150	150	100	100	....	....	....	....	....	....	....	....	....	100	200	250	250	150
Vitamin K	..	Oral 6 days		2																				
Decholin sod. 20% 10 cc.	..	..	..	1	1	2	....	....	....	....	....	....	....	....	....	....	....	....	....	1	2	1		

was 82 per cent. Both of these tests indicated only fair response of the liver to treatment. Kidney function was normal, the blood NPN was 30 mg. per cent, urea clearance 70 per cent. A 600 cc. transfusion of citrated whole blood was given the day before operation.

Cholecystotomy with drainage and an abdominal exploration was done under novocain spinal anesthesia. The liver was enlarged and mottled with purplish and yellow markings. The gallbladder was distended with approximately 16 ounces of green, watery fluid containing white sediment. This was aspirated. There were no stones. The common duct was thickened and approximately 1 inch in diameter. Palpation of the common duct and pancreas revealed nothing abnormal. One of several enlarged lymph nodes along the common duct was excised for microscopic examination. This proved to be inflammatory. Exploration of the remaining abdominal and pelvic viscera was negative.

The operative procedure was well tolerated with blood pressure maintained throughout. The postoperative course was uneventful for the first week. A blood transfusion (600 cc.) was given on the day after operation. Infusions of glucose were given daily for six days with 10 cc. of decholin sodium 20 per cent on the day of operation and on the first, second and fourth postoperative days. Decholin therapy was used to flush the infected biliary passages and to promote diuresis. (Table 1.) After the fourth postoperative day the temperature returned to normal where it remained for fourteen days.

Two hypodermic injections of vitamin K were given on the day of operation; a prothrombin value of 54 per cent was obtained on the first and again on the third postoperative days. The blood NPN was 33 mg. per cent. The hippuric acid response was reduced to 70 per cent and the icteric index was 22 on the third day after operation, indicative of adequate renal excretion but diminished liver function.

By the sixth postoperative day adequate nourishment and fluids were being taken orally, the parenteral glucose-decholin therapy was discontinued and bile salts (bilron) were given orally. Thereafter the patient gradually began to lose ground. By the end of the second week there was no desire for food. The cholecystotomy tube sloughed on the tenth day. Clear bile continued to drain through the gallbladder fistula which had formed. Intravenous glucose and decholin was started again on the sixteenth postoperative day, but despite an accelerated urinary output (Table 1) on the seventeenth and eighteenth days, the blood NPN continued to rise. A NPN of 110 mg. per cent was obtained on the seventeenth postoperative day (blood urea nitrogen 94 mg. per cent). The patient had become drowsy and when aroused appeared utterly exhausted.

The uremic coma deepened. On the nineteenth day the skin and sclera were definitely icteric. The pulse remained between 80 and 100 per minute and, although the circulation was sluggish, there was no evidence of cyanosis. Clear bile continued to drain from the fistula. The urine output became scanty (2 ounces in twelve hours) and contained red blood cells. Later an emesis of dark coffee grounds material occurred. The prothrombin was 22 per cent, the icteric index 22. The patient expired quietly in uremic coma on the twenty-first day after operation with complete suppression of urine.

At autopsy there was no free fluid in the abdomen. The liver appeared swollen, was firm and the surface was scarred with a number of irregular gray areas 1 to 3 cm. in diameter. In the dome of the left lobe was a larger irregular, dense area measuring 3 by 5 cm. The cut surface revealed liver parenchyma infiltrated with irregular strands of rubbery, gray tissue which would not scrape off. Microscopic examination showed these areas to be organized fibrous connective tissue. Sections from the liver showed atrophy of the hepatic cords in some areas while the cells of other areas appeared large and vesicular. There was moderate periportal lymphocytic infiltration and an increase in periportal connective tissue.

The pancreas, which felt cystic before removal, proved to have most of the parenchyma replaced by flabby connective tissue. Microscopically the pancreas appeared densely fibrous. Numerous islets of Langerhans remained intact.

The kidneys were average in size and flabby and the capsules stripped fairly easily. The cortex appeared beefy red, the calices and pelves normal. Microscopically the glomeruli stood out clearly and were engorged with red blood cells. There was cloudy swelling of the tubular epithelium without evidence of de-

generation. The tubules contained many red blood cells. Anatomic diagnosis: (1) biliary cirrhosis, acute hepatic degeneration, (2) pancreatic atrophy and fibrosis, (3) mesenteric lymphadenitis and (4) hemorrhagic nephritis.

Comment. This case is our best example of the hepatorenal syndrome as described by others. At no time either during or after operation was there a period of hypotension which would introduce the factor of anoxia. As long as parenteral therapy was continued the postoperative course seemed satisfactory. When this was withdrawn the clinical course headed progressively downhill. This may or may not have been coincidence. The 8 per cent drop in prothrombin level and a normal NPN immediately after operation indicates some depression of hepatic activity and no appreciable renal impairment. By the third day jaundice was apparent and the icteric index had increased to 22 in the presence of a normal blood NPN.

In view of the pancreatic fibrosis the possibility of external pancreatic secretion deficiency as a contributing factor to liver dysfunction is a point for speculation. The liver sections showed in addition to acute degenerative changes considerable fatty degeneration. It is conceivable in this particular patient that prolonged impairment of hepatic detoxication threw an unburdenable load on the kidneys. It is noteworthy that this patient showed none of the circulatory or central nervous system manifestations which characterized the postoperative course of the majority of patients in this series.

Case II. M. H., a female, age fifty, entered on the medical service complaining of gallbladder trouble and yellow jaundice. The present illness dated back four months during which interval there had been recurrent attacks of upper right quadrant, colicky pain, anorexia, epigastric fullness relieved by vomiting, jaundice and severe itching. There was a history of jaundice of nearly a year's duration in 1931 with symptoms comparable to those of the present attack. This cleared up and for three years the health was relatively good. In 1934 the patient had another series of attacks of knife-like, upper abdominal pain and jaundice. A course of "fruitola" was taken and after a night of cramping pains several large gallstones were recovered from the stool. These were in the patient's possession and were true gallstones of the calcium-pigment variety. The largest measured 15 mm. in diameter. Following the passage of the stones the symptoms and jaundice abated. Only minor gastrointestinal distress was experienced during the next six years.

During the four months preceding hospitalization there had been a weight loss of 40 pounds and the patient had become more irritable. The remainder of the history was irrelevant.

Physical examination revealed a poorly nourished, moderately jaundiced white female who appeared ill. The temperature was 99°F., pulse 90 and regular, blood pressure 115/65 mm. The abdomen was soft with marked tenderness and a questionable mass in the upper right quadrant. The remaining findings were not remarkable. A flat plate x-ray of the gallbladder area revealed nothing.

Laboratory data were as follows: Hemoglobin 70 per cent, red blood count 3,080,000, white count 16,150 with normal differential Routine urinalysis was negative A PSP test for kidney function gave 55 and 15 per cent for the first and second hours. Mosenthal test: specific gravity 1.005 to 1.017. The icteric index was 37.

The patient was observed and treated for two weeks with a high carbohydrate-low fat diet, daily infusions of 100 Gm. glucose, hot packs to upper abdomen and nitroglycerin sublingually for pain. Two 500 cc. transfusions of citrated blood were given. During this period the temperature fluctuated between normal and 100°F. and the icteric index declined from 37 to 21. An intravenous hippuric acid test was 46 per cent, the prothrombin time 116 per cent. The patient was transferred to the surgical service and was treated for an additional two weeks. Intravenous glucose was discontinued for four days and then resumed, 100 Gm. daily for ten days. The following additional therapeutic measures were instituted daily: glycine in fruit juice, liver extract orally, bile salts and hematinic plastules. An intramuscular injection of concentrated liver

extract was given daily for seven days. During this time normal prothrombin values prevailed; however, the hippuric acid test values declined progressively. (Table II.) The blood NPN was 30 mg. per cent. The patient had remained free of abdominal distress, except bloating. A faint icterus persisted, accompanied by itching of the hands and feet. A purpuric area approximately 2 by 3 inches appeared spontaneously on the left forearm. Bleeding, clotting and prothrombin times were normal. The platelet count was 174,000. The preoperative hippuric acid test remained critically low (22 per cent) indicating irreparable liver damage.

TABLE II

CASE II. M. H.

Days Pre- and Postoperative	31	28	26	23	19	15	13	10	4	0	1	2	3	4	5
Temperature (max.)	99°	100°	98°	100°	99°	100.2	99°	99°	98.6	98.6	99.6	99°	100.6	100.2	101
NPN, mg. %	..	..	..	..	..	...	..	30	..	....	....	51	....	60	
Hippuric acid test %	..	...	..	..	46	...	20	18	22	Operation					
Prothrombin %	..	..	..	..	..	116	..	102	..						
Icteric Index	37	30	23	23	33	20	..	...	21	....	....	86	....	120	
24 hr. fluid intake cc	..	..	..	..	..	...	..	...	..	3000	2000	3000	2000	2000	
24 hr. urine output cc	..	..	..	..	..	...	..	...	..	240	120	355	1350	1500	550
Bile drainage cc	..	..	..	..	..	...	..	...	..	400	300	480	320	120	
Decholin, 20%, 10 cc	..	..	..	..	..	...	..	...	..	1	1	1	1	1	
Parenteral glucose, Gm	..	..	..	..	..	...	..	...	..	300	200	150	150	150	

Laparotomy and gallbladder drainage under procaine spinal anesthesia was done thirty-one days after admission. The gallbladder was distended with white bile which was aspirated. Two large pigmented stones and cholesterol gravel were removed. Approximately one liter of cloudy ascitic fluid was aspirated from the abdominal cavity. The liver was subnormal in size, grey-blue in color, very hard and of hobnail appearance. The common duct was involved in adhesions and was left undisturbed because of the patient's poor condition. Catheter drainage of the gallbladder was provided. Fifteen minutes after the spinal anesthetic the blood pressure dropped to 86/54. Adrenalin by hypo, oxygen by mask and intravenous glucose solution relieved the hypotension promptly. Immediately after operation a transfusion of 500 cc. citrated whole blood was given.

During the twenty-four-hour period following operation the general condition seemed satisfactory; however, the urinary output was but 240 cc. from an intake of 3,500 cc. containing 250 Gm. glucose and 1 ampule decholin sodium (10 cc. 20 per cent). Throughout the remaining postoperative period 200 Gm. glucose and 1 ampule decholin sodium were given intravenously daily.

During the second twenty-four-hour period the output was reduced to 120 cc. from an intake of 2,000 cc. Three hundred cc. of bile drainage was collected during this period. Nasal oxygen which had been given twenty minutes of each hour since operation was discontinued. Forty-eight hours after operation the icteric index had become elevated to 86 and the NPN was 51, indicating impairment of both liver and kidney function. The patient was very restless and complained of severe itching. Nausea requiring gastric lavage every six to eight hours had developed. On the second postoperative day (third twenty-four-hour period) the patient seemed more alert and was less nauseated, but the itching continued and the jaundice was more apparent. The urinary output increased to 355 cc. On the third day 1,350 cc. of urine was excreted; however, the course continued downhill and the NPN continued to rise. Despite free bile drainage from the cholecystotomy tube and the increased urinary output which continued on the fourth day (Table II) the icteric index reached 120 and the NPN 60 mg. per cent. The patient had become very restless and was confused at times. Continuous gastric lavage was necessary. Persistent cyanosis despite oxygen therapy developed. The pulse and blood pressure could not be obtained before respiration ceased.

Comment. This case is a classical example of hepatorenal syndrome. Before

operation we were dealing with advanced hepatic insufficiency, at least in the function of detoxication, which could not be improved by specific therapeutic measures. Operation was followed by evidence of increased liver damage and renal decompensation with urinary suppression and azotemia. The degree of renal insufficiency was of such magnitude that, despite an induced diuresis on the third and fourth postoperative days, nitrogenous retention was progressive. The terminal picture was one of deepening icterus, uremia and circulatory collapse.

Case III. B. T., a female, age twenty-seven, was referred to the surgical service for splenectomy. The patient had contracted malaria at the age of six years which was apparently cured with quinine. The next thirteen years were uneventful. At the age of nineteen she suffered a severe case of Vincent's angina which resulted in the loss of most of the teeth and a swelling in the neck that required incision and drainage. Two years later the patient began to develop intermittent attacks of pain under the left costal margin. These attacks which occurred once or twice a year or oftener, were always followed by varying degrees of jaundice. She was hospitalized on two occasions over a three-year period with diagnoses of splenic anemia on one admission and chronic malaria on the other. Intramuscular atabrine was followed by improvement; however, during the next four years her health was not good. A change in climate was advised and she moved from Arkansas to Michigan a year before the present admission. For a few months she felt well and gained weight. Six months before admission abdominal swelling thought to be due to fluid developed. Several months later the patient was considerably weakened by loss of blood which was expelled by emesis and in the stool. Seven blood transfusions were given. Biopsy of liver and spleen was done at the University Hospital at Ann Arbor, Michigan, with a diagnosis of Banti's disease. Splenectomy was recommended. Paracentesis had been done on three occasions, the largest amount of fluid being 3 pints.

Physical examination revealed a restless, fairly well developed, emaciated, deeply jaundiced white female. Most of the teeth were missing. The essential findings were decreased area of liver dullness, an enlarged spleen which filled most of the upper left quadrant and a moderate amount of fluid within the abdominal cavity.

Laboratory data were as follows: Hemoglobin 75 per cent, red count 3,790,000, white count 4,250 with a relative lymphocystosis. Over a five-day period daily blood smears for malaria were negative. The platlet count was reduced to 55,040. Bleeding and coagulation time were normal. Fasting blood sugar was within normal limits. The hippuric acid test was 32 per cent, icteric index 120, serum protein 7.5 Gm., blood NPN 30 mg. per cent, and urea clearance decreased to 62 per cent. Routine urinalysis was negative except for one cast.

During five days of preparation for surgery the patient experienced two mild chills without fever. Preoperative treatment included intravenous glucose with thiamine and ascorbic acid, oral bile salts, liver extract and glycine.

At operation under novocain spinal anesthesia the spleen filled the upper left quadrant. The liver was small and uniformly hob-nailed. Splenectomy was done. There was a transient fall in blood pressure to shock levels that responded promptly to subcutaneously administered adrenalin. Oxygen-carbon dioxide was given by mask throughout the operation.

Immediately after operation a 500 cc. citrated blood transfusion and 1,000 cc. 10 per cent glucose were given. This was followed by 2,000 cc. 5 per cent glucose in water by hypodermoclysis. The twenty-four-hour urinary output from this intake was 200 cc. Blood pressure was maintained for the first eighteen hours and then declined to 64/?. The pulse rate was 140 per minute. The patient was stuporous. Following 500 cc. plasma-glucose and 500 cc. 20 per cent glucose with 1 ampule of decholin the blood pressure increased to 110/60 with a pulse rate of 112. Three hours later the patient was semicomatose with a blood pressure of 60/25 and an apex rate of 160. Oxygen by nasal catheter was begun and 500 cc. of citrated blood by vein elevated the blood pressure to 90/30. The pulse rate was 140. The patient was comatose. The eyes were open but there was no response. During the second twenty-four hour period the urinary output was 220 cc.; however, only 40 cc. were obtained during the last ten hours of the period.

Forty-eight hours after operation the patient was in coma and was deeply jaundiced. The blood pressure was 77/?, apex rate 160. Five hundred cc. of plasma and 500 cc. 20 per cent glucose with calcium gluconate raised the blood pressure to 110/50. Over a six-hour period 90 cc. of urine was excreted. Within two hours after the plasma infusion the blood pressure declined to 90/40 with a pulse rate of 154. Spells of muscular twitching which developed into clonic convulsions lasting two to three minutes followed. The temperature, which had fluctuated between 100° and 101°F. during the preceding twenty-four hours, climbed to 105°F. and the patient expired quietly in circulatory collapse.

Comment. This patient, like Case II, had advanced portal cirrhosis with significant hepatic insufficiency as measured by the hippuric acid test. Portal cirrhosis is characterized pathologically by patchy involvement of hepatic parenchyma and physiologically by relatively well maintained hepatic function until the disease is well advanced. Areas of connective tissue replacement alternate with areas of normal or even hyperplastic hepatic cells. Because of the large factor of safety possessed by the liver only a fraction of normal parenchyma can maintain hepatic function. On the other hand, diffuse hepatic lesions which compromise the entire parenchyma give evidence of diminished function early.

Both Cases II and III gave laboratory evidence of depleted hepatic reserve before operation. It is not unlikely that the kidneys in these patients were subjected to toxic products which would have been rendered inocuous by a normal liver. The profound circulatory instability exhibited by Case III was an outstanding feature of the postoperative course.

2. *Renal Insufficiency with Hepatic Insufficiency.* Case IV. F. G., a female, age sixty-four, was admitted to the hospital five months after permanent drainage of the gallbladder elsewhere for a partial common duct obstruction. The drainage tube had been clamped off most of the time, but on occasions there were spells of upper right quadrant distress with nausea. These were always relieved by unclamping the tube to the gallbladder which was followed by drainage of several ounces of bile. Following the first operation at which time the patient was deeply jaundiced much of her former vitality had been regained. Of late there had developed anorexia, loss of strength and foamy, clay-colored stools off and on. The intervals between spells of upper abdominal distress had diminished progressively to every few days.

Except for attacks of gallbladder colic for two years prior to the onset of painful, complete obstruction of the common duct which led to the above operative procedure, the past history was irrelevant. There were no symptoms referable to the cardiovascular and renal systems.

Essential physical findings revealed an elderly white female who appeared somewhat malnourished and whose skin had a leathery, brownish-yellow tinge. The blood pressure was 122/70, the pulse 64 and regular. Heart and lungs were negative. A rubber drainage tube protruded through an indurated, healed upper right quadrant scar.

Laboratory findings on admission were as follows: Hemoglobin 89 per cent, red blood count 4,300,000, white blood count 8,750. Bleeding time two minutes, coagulation time four minutes, prothrombin time 89 per cent. Blood NPN 30 mg per cent, urea N 12 mg. per cent, serum protein 5.7 Gm. The urea clearance test showed impaired kidney function (55 per cent). Icteric index was elevated to 24, fasting blood sugar was 92 mg. per cent and blood amylase at the lower limits of normal (66 units, Somogyi). A subnormal hippuric acid test of 72 per cent on admission was raised to 89 per cent following six days of preparation for operation. With the bromsulfalein test, 40 per cent of the dye was retained after thirty minutes.

Following lipiodol injection of the biliary tract through the cholecystotomy tube which produced typical gallstone colic pain a transient elevation of temperature (102°F.) and an elevated blood amylase was observed (242 units). After five days of therapy, designed to build up hepatic reserve and combat bile salt and pancreatic deficiency, exploration of the common duct under spinal anesthesia was done. Much débris and two faceted stones were removed from the ampulla and T-tube drain-

age of the common duct was provided. The operative procedure was tolerated well.

For the first seventy-two hours after operation the urinary output was diminished. (Table III.) On the first postoperative day the blood pressure declined to 82/54. Following hepatic damage contributed to the clinical picture. On the morning of the sixth postoperative day the NPN dropped to 84 mg. per cent and during this and the following day the twenty-four-hour urinary output was 2,000 cc. Clinical improvement followed and gastric

TABLE III
CASE IV. F. G.

Days Pre- and Postoperative	7	4	1	0	1	2	3	4	5	6	7	8	9	10	11	12	13
Temperature	98°	99°	97.8	98°	99°	98°	98.4	98.4	98.4	98.4	98.4	98.6	98°	98°	98.8	98.4	98.4
NPN mg. %	30	...	...	....	....	88	102	116	128	84	65	....	56	50	47	42	34
Urea clearance %	55	...	...	....	....	....	12										
Hippuric acid %	72	...	89														
Prothrombin %	89	...	...	....	....	80	....	....	90								
Icteric index, units	24	...	...	....	....	16	....	....	....	....	....	....	....	....	....	....	9
BSP test, %	40	...	...	....	....	0											
Fluid intake, 24 hr./cc.	..	...	...	2500	2500	3000	3000	1800	2000	3800	2750	3000	2200	1500	2000		
Urine output, 24 hr./cc.	..	...	...	300	750	500	1870	920	1500	2000	2000	1580	820	820	1420	1110	800
Parentenal glucose, Gm.	..	100	100	200	200	300	250	200	200	200	100	100	100	....	100		
Decholin, 20%, 10 cc.	..	...	...		1	1	1	1	1	1	1	1	1	....	1		

intravenous fluids, circulation improved and for the next fourty-eight hours the pressure varied between 108/70 and 122/74 mm. The pulse rate never exceeded 90 per minute. It was necessary to provide continuous gastric lavage which, however, did not allay spells of singultus. Intravenous decholin sodium was started on the first postoperative day and was continued along with intravenous and subcutaneous glucose for nine days. (Table III.) On the second postoperative day there was no retention of bromsulfalein thirty minutes after injection of the dye and the prothrombin time was on 9 per cent under the preoperative value, indicating no appreciably increased impairment of hepatic activity as reflected in these two hepatic functions. A blood NPN of 88 mg. per cent and a urea N of 48 mg. per cent suggested rather marked impairment of kidney function. A progressive azotemia developed in spite of increasing urinary output and the clinical picture was that of uremia. The greatest nitrogen retention occurred on the fifth postoperative day (NPN 128 mg. per cent, urea N 80 mg. per cent). At this time the prothrombin value was 90 per cent of normal. Inasmuch as this corresponds to the preoperative prothrombin level and since no vitamin K had been administered postoperatively, it seems unlikely that any significant degree of generalized lavage was discontinued on the seventh postoperative day. Thirteen days after operation the nitrogenous constituents of the blood and icteric index were normal and the patient recovered rapidly.

Comment. This patient is one in whom, in view of the facts related to the biliary tract disturbance, one would anticipate a narrow margin of hepatic reserve. The clinical picture after surgery conforms with that frequently classified as "hepatorenal," yet two hepatic functions (prothrombin and dye removal) gave no indication of hepatic insufficiency on the second postoperative day. From blood chemistry and urinary output data the azotemia which developed would seem most satisfactorily explained on the basis of impaired kidney function due to renal disturbance. The impaired urea clearance before operation favors this view.

3. *Associated Hepatic and Renal Insufficiency.* CASE V. R. S. M., a male, age seventy-six, entered the hospital with a suprapubic catheter in the bladder. Eleven days previously the patient had experienced acute urinary retention due to benign prostatic hypertrophy which had caused some symptoms for a year.

The patient was almost deaf and a detailed history was not obtained; however, it was learned from the family that the patient's general health had always been good and there had been no serious illnesses or gastrointestinal symptoms. There was occasional dizziness and moderate dyspnea on exertion.

Physical examination revealed a well preserved, elderly white male who was alert and not acutely ill. The pulse rate was 76, respiration 18, blood pressure 120/70 mm. The teeth were in poor condition, the heart and lungs seemed normal, the abdomen was soft without masses or tenderness, the suprapubic incision was well healed. By rectal examination the prostate was of the size, shape and consistency of a baseball.

Laboratory data were as follows: Hemoglobin 80 per cent, red blood count 4.23 million, white blood count 5,050 with a normal differential; urine, specific gravity 1.020 with a trace of albumin and many pus cells. Blood NPN 42 mg. per cent, urea nitrogen 22 mg. per cent, and urea clearance 58 per cent, indicating impaired kidney function. Prothrombin was 92 per cent and there was no retention of bromsulfalein in thirty minutes, indicating no impairment of liver function.

The bladder infection was treated for one week with sulfanilamide and 500 cc. infusions of 20 per cent glucose with 10 cc. decholin sodium 20 per cent were given daily for seven days to promote diuresis. Following this, decholin tablets (twelve daily) were given for four days. The blood NPN which reached a maximum elevation of 68 mg. per cent two days after beginning the intravenous therapy declined to 39 mg. per cent ten days later.

At operation suprapubic prostatectomy was done without difficulty under novocain spinal anesthesia. Three hours after operation the blood pressure was 70/56, the pulse rate 100 and rather weak. The dressings were soaked with sanguinous drainage but there was no evidence of undue hemorrhage. Following the intravenous injection of 500 cc. 20 per cent glucose with decholin, ephedrine by hypodermic and oxygen by nasal catheter the blood pressure rose to 115/70. Five hours later the pulse was weak and thready and the blood pressure could not be obtained. A 400 cc. citrated whole blood transfusion was without effect on the low blood pressure. The pulse was somewhat irregular. Two hypodermic injections of ephedrine (50 mg.) and two ampules of digitalis given an hour apart elevated the blood pressure to 76/42. An ampule of pitressin by hypodermic raised this to 94/64. Fourteen hours after operation the patient appeared slightly jaundiced and during the night there were several emesis, the last of which was blood tinged. The pulse remained poor in quality. Twenty-four hours after operation the patient was semicomatose, there was moderate jaundice, a blood pressure reading could not be obtained, and a gastric lavage contained coffee ground material. Continuous gastric drainage was instituted. Following 500 cc. of plasma-glucose and 500 cc. 20 per cent glucose with decholin the blood pressure rose to 150/85, the pulse became stronger and the patient looked and acted greatly improved. This state was maintained throughout the first postoperative day. The blood NPN was 70 mg. per cent. The urine output was 400 cc. for the first twenty-four hours and 1,300 for the second twenty-four-hour period. Thereafter permanent urinary suppression set in. On the second postoperative day the temperature was 99°F, the blood NPN was 108 mg. per cent, there was singultus at intervals and cyanosis was apparent in spite of continuous oxygen therapy. Fluid intake was maintained parenterally. On the evening of the second day the patient was stuporous, the icterus was more pronounced (icteric index 80), the blood pressure was 70/?, the pulse rate 130 and poor in quality.

On the third postoperative day the temperature reached 101°F, the blood NPN was 135 mg. per cent, singultus continued, the pulse was 144 and thready and the blood pressure could not be obtained except for several hours following intravenous infusions. On the morning of the fourth day the patient was in deep coma, there were moist râles throughout the lungs and the skin and sclera were deeply jaundiced. The blood pressure could not be obtained. The temperature increased to 104°F and the patent expired ninety-six hours after operation.

At autopsy the skin was deep yellow. The abdominal cavity appeared normal. The prostatic bed contained an adherent blood clot. The liver was larger than average, the borders were rounded and the surface appeared dusty yellow. The cut surface had a greasy appearance and felt granular when scraped. Microscopically the liver cords were shrunken throughout; in many areas the normal pattern

was lost with the hepatic cells appearing large and vesicular and arranged in clumps.

The kidneys were small, the surfaces scarred and the capsules adherent. Cut surface revealed narrowing of the cortex with indistinct markings. Under the microscope the glomeruli were shrunken and their epithelium showed advanced sclerotic changes. The cells of the tubules were swollen and clear; in some areas the protoplasm took almost no stain. Desquamation and other advanced degenerative changes in the tubular epithelium were scarce.

The lungs were wet but contained no areas of consolidation. The remaining findings were irrelevant.

Comment. This case illustrates a combination of acute hepatic degeneration followed promptly by renal failure in a patient with a normal NPN but no renal reserve. The liver damage accompanied by jaundice within twelve hours of operation may or may not be related to the pronounced tendency to surgical shock. The absence of fever during the first forty-eight hours after operation makes the possibility of a blood stream infection unlikely. The vasomotor instability was the most outstanding clinical feature of the postoperative course.

Decholin therapy before operation produced a diuresis but the elimination of urinary nitrogen (twenty-four-hour periods) was not increased, suggesting that any benefit from decholin in azotemia results from some mechanism other than increased urinary excretion of nitrogen bodies.

The following case is interesting both from a diagnostic standpoint and in its postoperative hepatorenal aspects:

Case VI. F. J., a white female, age forty-nine, was admitted to the medical service and transferred to the surgical service a week later. Three weeks prior to admission the patient had an attack of what was thought to be the "flu." There was generalized muscular aching with loss of appetite but, it was believed, no fever. The patient remained in bed for two days but did not regain her former strength or appetite.

One week after the above illness the patient's family noticed that her eyes and skin were turning yellow. The patient denied any abdominal distress or attacks simulating colic. The appetite had been only fair, and for several years there had been occasional spells of gas and bloating. The bowels had been costive as a rule for a number of years, but there had been no noticeable change in bowel habit and no bloody or tarry stools. The remaining past history was irrelevant except that her father died of carcinoma of the liver.

Physical examination revealed nothing of diagnostic value. The patient was alert, intelligent and cooperative. There was an icteric tint to the skin and sclera, the lungs and heart appeared normal, the blood pressure was 165/100, the pulse 80 and regular. No abdominal masses or tenderness were made out.

A preliminary diagnosis of catarrhal jaundice was made. An intravenous hippuric acid test of 14 per cent and a prothrombin value of 76 per cent that declined to 45 per cent four days later was thought to substantiate the diagnosis of intrahepatic jaundice. However, flat plate x-ray of the gallbladder region revealed one large and two smaller positive shadows and following three days of vitamin K therapy (kayquinone) with desoxycholic acid* orally, the prothrombin time promptly rose to 90 per cent of normal and the diagnosis was changed to obstructive jaundice, probably due to a silent common duct stone. This diagnosis seemed further strengthened by the fluctuating icteric index during nine days of observation (Table IV.)

The patient responded promptly to a high carbohydrate high protein diet, bile salts and vitamin K and intravenous glucose, and before operation the prothrombin was normal and the hippuric test (intravenous) was 70 per cent. The blood NPN was 30 mg. per cent, urea clearance decreased to 45 per cent (25 per cent below the considered normal of 70 per cent), hemoglobin 77 per cent, red blood count 3.73 million, white blood count 5,550 with normal differential and the urine negative except for the presence of bile. The icteric index had increased to 150; quantitative blood bilirubin was 12.0 mg. per cent (normal 0.2 to 0.4 mg. per cent).

Operation was performed under novocain spinal anesthesia. The gallbladder and common

* Decholin sodium and desoxycholic acid (degalol) used with vitamin K for differentiation of intra- and extrahepatic jaundice on the basis of prothrombin response has been kindly supplied by Riedel de Haen and Co., New York.

duct were only questionably distended and the contained bile was pale green and thick with mucus. The common duct was explored and found to contain one pea-sized stone. Several stones were removed from the gallbladder and tube drainage was provided for both the gallbladder and common duct. Exploration of the pancreas and gastrohepatic omentum revealed in the latter a hard nodule the size of a hazelnut which was taken for microscopic examination. The upper surface of the liver was smooth; however, palpation of the under surface revealed multiple hard nodules beneath the capsule and surrounding the bifurcation of the common bile duct. The lower abdomen was then explored and a hard, circumscribed neoplasm was found in the large bowel just distal to the splenic flexure.

TABLE IV

CASE VI. F. J.

Days Pre- and Postoperative	8	6	3	2	1	0	1	2	3	4	5	6	7	8	9	10	11	12	13
Temperature	98[1]	98[4]	98[4]	98[4]	98[4]	98[4]	99[2]	100[2]	100[2]	98[1]	98[0]	99[0]	98[4]	98[2]	98[2]	98[0]	98[8]	97[6]	98[4]
NPN mg. %	..	....	...	...	30	....	....	....	72	76	88	110	110	....	106	114	136	136	
Urea clearance %	..	....	...	...	45														
Hippuric acid %	14	....	42	...	70														
Prothrombin %	76	45	...	80	90	....	....	90	65	58	80	84	90	70	52	40			
Icteric index, units	32	20	110	100	150	....	200	....	....	220	....	250	....	....	....	250			
Fluid intake, 24 hr./cc	..	....	...	...	...	3400	3750	2000	4200	3500	1500	1500	1500	2500	2500	1250	1800	1000	
Urine output, 24 hr./cc	..	....	...	...	...	100	80	150	40	20	65	0	25	30	45	60	40	65	
Parenteral glucose, Gm	..	....	...	...	...	200	150	200	250	250	150	200	150	200	100	150	150	150	
Vitamin K-bile salts—oral*	..	4 days																	
Vitamin K-hypo	..	....	...	...	...	....	....	....	....	1	1	1	1						
Decholin, 20 cc. 10%	..	....	...	...	...	....	1	2	1	1									

* Kayquinone Abbott 75 mg. and Degalol (Reidel de Haen) 18 gr.

During the operative procedure there was a transient fall in blood pressure from 125/84 to 80/20. Adrenalin, 0.5 cc. by hypo and 95 per cent oxygen-carbon dioxide 5 per cent by mask properly restored the pressure to 140/100; during the following thirty minutes this declined slowly to 100/78 where it was maintained.

Twelve hours later the patient was resting quietly, aroused easily and was taking fluids well. The blood pressure during the first twenty-four-hour period was maintained at 100/65, the pulse rate 120 and regular, the urinary output 100 cc. and from this time on an almost complete suppression of urine ensued. (Table IV.) There was likewise essentially no bile drainage. Hypertonic glucose and decholin were without effect on the anuria. On the fourth postoperative day it was necessary to institute continuous gastric drainage; however, the patient's general condition was amazingly good considering the almost complete blockage of both liver and kidneys. The blood NPN had risen to 76 mg. per cent, and the prothrombin had dropped from 100 per cent on the second postoperative day to 58 per cent. Vitamin K was given by hypodermic injection for four consecutive days and a prompt response in prothrombin time to 90 per cent was observed on the seventh postoperative day. Following withdrawal of vitamin K therapy the prothrombin value declined progressively to 40 per cent.

By the eleventh postoperative day the patient was dull and exhausted with spells of nausea and singultus. The breath had a uriniferous odor and the blood NPN was elevated to 136 mg. per cent. Except during the first twenty-four hours postoperatively, the blood pressure remained above 120 mm. systolic and the temperature was essentially normal. Clinically, death on the thirteenth postoperative day appeared to be due primarily to uremia.

Postmortem examination revealed complete walling off of the gallbladder and common duct region with omental adhesions. There was no evidence of bile or infection in the abdominal cavity. The liver showed gross and microscopic evidence of early obstructive biliary cirrhosis with distended bile capillaries, increased pigmentation and varying degrees of central atrophy of liver cells. Microscopically the nodules in the liver hilus and that removed at operation from the gastrohepatic omentum showed the same metaplastic cellular structure interpreted as metastatic from the adenocarcinoma in the large bowel. The kidneys were small and scarred, the capsules stripping with difficulty. The microscopic picture was that

of fairly advanced chronic glomerulonephritis with varying degrees of hyaline degeneration of the glomeruli and atrophy of many of the collecting tubules. Pigmented débris and degenerative cellular changes were prominent throughout the tubular areas.

Comment. This case is extremely interesting in that observations were made over a thirteen-day period during which there was essentially complete obstruction to the liver and an almost complete suppression of urine. During the first half of this period the general condition appeared unbelievably good under the circumstances. It is noteworthy that during this period the obstructed liver still responded promptly to vitamin K therapy, a response not obtained in the presence of intrinsic rather than extrinsic jaundice. It seems unlikely that a rapidly failing liver would manufacture prothrombin so promptly. On the basis of this response it would seem that despite the clinic resemblance to the hepatorenal syndrome, hepatic insufficiency played little if any part in the marked urinary suppression observed during the first seven or eight days following operation. On the other hand, the degree of chronic glomerulonephritis present undoubtedly narrowed significantly the degree of renal reserve. Although there was no nitrogenous retention before surgery, the lowered urea clearance test (45 per cent) is evidence of a lowered renal factor of safety. The following explanation is offered to account for the course of events which followed surgery in this patient:

The transient fall in blood pressure during operation and the relatively low pressure for the next twelve to sixteen hours was sufficient in this particular individual, whose original blood pressure was 165/100, to produce a lethal anoxia in the remaining functional kidney parenchyma. Degenerative changes resulting from this damage, coupled with the added load of bile excretion, was sufficient to plug the remaining functional collecting tubules to the extent that the excretion of urine was reduced almost to nothing. (Table IV.)

Case VII. The patient, H. B., a female, age sixty, was transferred from the medical service with a diagnosis of acute cholecystitis with lithiasis and obstruction of the common duct, partial. The past history was one of gas and bloating with belching and burning in the epigastrium of fifteen years' duration. Eight days before admission the patient had an attack of severe gallbladder colic requiring several hypodermic injections of morphine for relief. Following this marked jaundice developed. On admission the icteric index was 100, blood NPN 32.79 mg. per cent and the hippuric acid test 71 per cent. Surgical intervention was refused and after eleven days of medical management the patient was dismissed on a high carbohydrate diet and bile salts. On dismissal the icteric index had declined to 75.

The patient was readmitted on the surgical service two weeks later. The jaundice had become more intense, dyspepsia for all types of food had developed along with recurrent attacks of upper right quadrant pain associated with distention and vomiting.

Essential physical findings revealed an obese, white female with yellow sclera and a canary yellow skin. There were no positive chest findings, the blood pressure was 130/80, the pulse 64 and regular. The abdomen was domed and tympanitic, there was marked tenderness without rigidity in the upper right quadrant and epigastrium, and a smooth tender mass the size of an orange was palpable under the right costal margin. The temperature was 98°F.

Laboratory findings were as follows: Hemoglobin 95 per cent, red blood count 4.84 million, white blood count 11,500 with an increase in segmented forms. Mosenthal test 1.004 to 1.016, NPN 30 mg. per cent, urea clearance test 56 per cent (subnormal). The prothrombin time was 62 per cent of normal. The icteric index was 200. During three weeks the hippuric acid test had dropped from 71 to 31 per cent.

The patient was placed on a high protein, high carbohydrate diet with Karo syrup in fruit juice, bile salts (bilron) and 1,000 cc. of 10 per cent glucose with 1 Gm. of calcium gluconate, 6 mg. of thiamine chloride and 100 mg. of ascorbic acid intravenously daily. Nitroglycerin, gr. $\frac{1}{150}$ sublingually and atropine sulfate, gr. $\frac{1}{75}$, was given three times daily on alternate days. The response was poor due to nausea and vomiting whether food was taken or not. It was decided that gall-

bladder drainage offered the only chance for improvement.

Under local novocain infiltration anesthesia the abdomen was opened by an upper right gridiron incision. The liver appeared enlarged with borders rounded, greyish in color, and mottled with yellow spots the size of a pin head. The gallbladder was distended to about four times average size; aspiration yielded 6 ounces of colorless fluid. Upon excising the dome of the gallbladder several ounces of dark green, mucoid material loaded with gravel was obtained. A number of stones were removed from the gallbladder and cystic duct and a rubber drainage tube was fixed in the gallbladder with purse-string suture. The common duct which was markedly distended was not disturbed. There was no evidence of metastases along the course of the common duct.

The operative procedure, which required thirty-two minutes, was well tolerated except for a persistently low blood pressure (70/40 to 90/60 mm.); 95 per cent oxygen-5 per cent carbon-dioxide was given by mask throughout the operation. At the end of the procedure clear, pigmented bile was draining from the cholecystotomy tube.

Following operation oxygen was given by mask (6 liters per minute) twenty minutes of each hour for the first twenty-four hours. Postoperative temperature, laboratory tests, fluid intake and output and volume of bile drainage are shown in Table v.

During the first twenty-four hours the blood pressure varied between 70 and 80 mm. systolic. One hundred sixty-five cc. of urine were obtained following fluid intake of 3,225 cc. containing 200 Gm. of glucose. At the end of this period the blood pressure was 105/65, the pulse rate 90, respiration 18. The general appearance seemed satisfactory and oxygen inhalations were discontinued. For the next twenty-four hours the patient was anuric in spite of an intake of 3,500 cc. The urinary output for the second twenty-four hour period was 90 cc. There was free bile drainage; however, the bile salt concentration for the first twenty-four hours was 380 mg. per cent (normal around 2,000 mg. per cent). Nausea and occasional emesis persisted, the jaundice appeared to be increasing, the blood pressure remained around 100 mm. systolic, the pulse rate varied between 70 and 88 and there had been no elevation of temperature.

Following hypertonic glucose and decholin (Table v) the urinary output increased to 275 and 270 cc., respectively, on the second and third postoperative days. The icteric index was 200, the blood NPN had increased to 50 mg. per cent. On the fourth and fifth postoperative days the urine output was increased to 930 and 1,030 cc. It was hoped that with improved renal elimination some clinical improvement might follow. Such was not the case. The blood pressure declined to between 70 and 80 mm. systolic and the patient appeared weakened and semistuporous. A 600 cc. citrated whole blood transfusion produced no change in the general condition or blood pressure level. For the last twenty-four hours of

TABLE V

CASE VII. H. B.

Days Pre- and Postoperative	6	5	4	3	2	1	0	1	2	3	4	5	6	7
Temperature	99^{2}	98^{6}	98^{0}	98^{4}	98^{4}	98^{8}	98^{0}	98^{4}	98^{0}	98^{0}	97^{4}	98^{6}	97^{2}	97^{0}
NPN mg. %	..	30	..	..	..	..	....	....	50	....	....	....	90	
Urea clearance %	..	..	56											
Hippuric acid %	..	..	..	31										
Prothrombin %	..	..	..	62	..	..	....	....	43	....	....	....	30	
Icteric index, units	200	..	..	170	..	..	....	....	200	....	....	....	200	
Bile drainage, cc	..	..	..	..	..	..	200	200	550	625	525	850	350	
Cholates in drainage bile, mg. %	..	..	..	..	..	..	....	120	....	100	....	....	86	
Fluid intake, 24 hr./cc	..	..	..	..	..	..	3225	3500	2500	2270	2000	1500	1500	
Urine output, 24 hr./cc	..	..	..	..	..	..	165	90	275	270	930	1030	90	
Parenteral glucose, Gm	..	..	..	..	..	..	150	200	150	200	100	....	100	
Decholin, 20%, 10 cc	..	..	..	..	..	..	....	....	2	1	1			

life the patient was in deep coma, and during twenty hours prior to death no urine was eliminated. The blood NPN was 90 mg. per cent on the sixth postoperative day. A free drainage of highly pigmented bile continued to the end, but on the sixth postoperative day the concentration of bile salts was critically low (86 mg. per cent).

Comment. This patient presented early hepatic insufficiency with delayed renal failure. Here again we encountered a very early oliguria which was later overcome for forty-eight hours. The hypotension of the first twenty-four hours following operation may have been responsible for the transient urinary suppression. If this was a typical case of hepatorenal syndrome from the onset with renal insufficiency due to toxic substances of hepatic origin, how can one account for the urinary output of 930 and 1,030 cc. on the fourth and fifth postoperative days? The persistently elevated icteric index in spite of free biliary drainage, and the progressive decline in bile salts in the drainage bile speak for markedly impaired hepatic function. The rising NPN and urinary suppression are indicative of renal dysfunction, possibly functional at first but later associated with peripheral vascular failure, organic in nature. Whether some specific toxic substance finally broke down kidney reserve by direct effect, or whether the latter occurred secondary to persistent hypotension and histotoxic anoxia is a point for speculation. A normal temperature was obtained throughout the illness. Unlike Cases III and V, the vasomotor instability was not associated with tachycardia.

Case VIII. M. S. M., a female, age sixty-two, developed jaundice and itching three days after an attack of gallbladder colic which recurred at intervals for two days. When admitted to the hospital the stools were clay colored, the icterus index was 97 but there had been no recent upper right quadrant pain. Hemoglobin was 74 per cent, red blood count 3,840,000, white blood count 7,000. The NPN was 34 mg. per cent, urea clearance slightly diminished to 60 per cent. An hippuric acid test of 23 per cent indicated marked liver insufficiency; however, a prothrombin time of 83 per cent was evidence against generalized liver damage. During five days of preoperative preparation for surgery there was no elevation of temperature and the patient remained free of distress. One hundred Gm. of glucose were given intravenously daily for three days before surgery. An hippuric acid test of 68 per cent the day before operation indicated a response to therapy.

Cholecystectomy and removal of a common duct stone with T-tube drainage of the common duct was done. The operation was tolerated well except for a transient fall in blood pressure to 94/70 with spontaneous return to 135/86. During the last twenty minutes of the operation 95 per cent-5 per cent oxygen carbon dioxide was given by mask.

On the evening of the day of operation the patient could not be roused for several hours following $\frac{1}{20}$ gr. dilaudid and $\frac{1}{200}$ gr. hyoscine. The color remained good, the pulse was 100 with an occasional dropped beat, the blood pressure 100/70 mm. Several hours later the effects of the sedative had worn off and the patient was easily aroused. Table VI shows fluid intake and output, temperature curve, blood chemistry determinations and the concentration of bile salts in the T-tube drainage. Eight hours after operation marked oliguria developed that persisted for forty hours. Between the sixteenth and twenty-fourth postoperative hours only 15 cc. of urine was excreted. During the second twenty-four-hour period the output was restricted to 60 cc., and the output for the third twenty-four-hour period was 66 cc.

On the morning of the second postoperative day the appearance was satisfactory, the patient was quiet, the temperature was 100.8°F., the blood pressure 140/86, the pulse 100 and regular. The bile drainage was scanty and dark containing only 120 mg. per cent of cholates. Prothrombin had diminished to 60 per cent, also indicative of suppressed liver function. The blood NPN was elevated to 58 mg. per cent. Decholin sodium was added to the intravenous glucose daily in quantities shown in Table VI. On the afternoon of the second day the patient appeared catatonic, staring at the ceiling and totally ignoring the examiner. The upper extremities were spastic but the reflexes were

equal and no pathologic reflexes were elicited. Later the appearance was more comatose, the breathing deep and labored, the blood pressure 140/90, the pulse 112 and regular.

The patient had been anuric for twelve hours. Continuous gastric lavage was started.

There developed a progressively deepening coma with muscular twitching and carpopedal spasm at intervals. Cheyne-Stokes respiration developed and there was some irregularity of the heart beat. Respiration ceased two minutes before the heart stopped.

TABLE VI

CASE VIII. M. S. M.

Days Pre- and Postoperative	5	3	2	1	0	1	2	3	4	5	6	7	8	9
Temperature	98^6	98^6	98^6	98^6	98^6	100^0	100^7	100^0	99^0	100^0	101^0	101^8	102^2	102^0
NPN mg, %	34	...	...	...	....	....	58	76	....	....	....	....	100	
Urea clearance %	60													
Hippuric acid %	23	...	...	68										
Prothrombin %	83	...	...	85	....	....	60	....	23	....	....	85		
BSP retention, 15 min. %	..	...	...	0	....	....	....	....	....	....	....	25		
Cholates in drainage bile mg. %	..	...	...	...	520	200	120	104	40	540	840	130	20	trace
Fluid intake, 24 hr./cc	..	...	...	...	4000	3500	4200	2500	3750	2000	3000	4500	2500	1000
Urine output 24 hr./cc	..	...	...	...	280	60	66	450	360	220	360	430	160	60
Parenteral glucose, Gm	..	100	100	100	300	350	400	250	250	300	200	400	250	100
Liver extract* 10 cc. (H)	..	...	...	...	....	....	2	2						
Vitamin K, hypo	..	...	...	...	....	....	....	1	1	1	1			
Decholin, 20%, 10 cc	..	...	...	...	....	2	1	1	1	1	1	1		

*Lilly, crude.

On the morning of the third day there was no appreciable change in the general condition. Several ounces of urine were obtained for the night and during the twenty-four-hour period the output rose to 450 cc. The blood NPN was 76 mg. per cent. By the fourth day the concentration of cholates in the bile (400 cc. of bile for twenty-four hours) was reduced to the critically low level of 40 mg. per cent, the prothrombin had dropped to 23 per cent and the icterus had increased (blood bilirubin 10.2 mg. per cent in contrast to 7.6 mg. per cent before operation). The gastric drainage contained coffee-ground material. A total of 20 cc. of liver extract was given intramuscularly on the third and fourth days. Vitamin K (hyquinone) was given by hypo on the fifth, sixth, seventh and eighth days and the prothrombin time rose promptly from 23 to 85 per cent on the seventh day. On the fifth and sixth days the cholates in the bile drainage increased to 540 and 840 mg. per cent, respectively, only to drop off again to 130 and 20 mg. on the seventh and eighth days. A bromsulfalein test on the seventh day showed 25 per cent retention in fifteen minutes and the blood amylase was elevated to 304. On the eighth day the blood NPN was 100 mg. per cent and the urea N 56 mg. per cent. (Table VI.)

Comment. Although renal and hepatic function tests gave evidence of subnormal function before operation, clinically this patient appeared a fairly good operative risk. Urinary suppression developed eight hours after operation. The oliguria was associated with definite hepatic insufficiency as reflected by the increasing icterus, the drop in prothrombin and the marked impairment of bile salt synthesis. Decholin sodium was without recognizable effect on the course of subnormal liver function. It may have been a factor in the increased urinary output which began twenty-four hours after beginning this therapy.

The increased concentration of bile salts in the T-tube drainage on the fifth and sixth days following parenteral liver extract denoted greatly improved hepatic synthesis as did the response in prothrombin formation following parenteral vitamin K. (Table VI.) Despite these favorable signs, the patient did not show any clinical improvement. The catatonic stupor and the manifestations of alternating stimulation and depression of the central nervous system was out of all proportion to the degree

of azotemia present. The postoperative temperature curve is very suggestive of an associated infection. Permission for autopsy was denied.

Case IX. M. S., a female, age fifty-three, entered the hospital with a history of recurrent attacks of severe colicky upper right quadrant pain with back and right shoulder radiation for twenty-five years. The initial attack in 1916 was described as severe. Between 1916 and 1939 the distress was milder in character, occurring at irregular intervals. During the past two years there had been an almost constant dull aching pain under the right costal border radiating at times to the right shoulder and to the epigastrium. There had been no jaundice, no fever or chills or weight loss. There was a selective dyspepsia for fried and "rich" foods.

Inventory by symptoms and past history were essentially negative except for the digestive disturbance. The patient had scarlet fever as a child. There had been four term pregnancies with normal deliveries and menopause in 1938.

Physical examination revealed a rather thin, fairly well nourished middle aged female with no remarkable physical findings except upper right quadrant tenderness. The blood pressure was 142/86, the pulse 88 and regular. Urinalysis was negative, hemoglobin 83 per cent, red blood count 4,060,000, white blood count 5,450 with a relative lymphocystosis. X-ray of the gallbladder after dye showed a poorly visualizing gallbladder with negative shadows.

Preparation for surgery for one week included in addition to a high caloric, high carbohydrate diet, vitamin B complex, bile salts and pancreatin. Atropine sulfate orally and luminal kept the patient free of discomfort.

Table VII
CASE IX. M. S.

Days Pre- and Postoperative	6	3	0	1	2	3	4	5	6	7	8	9	10	12	13
Temperature	98^4	98^6	98^8	101^0	100^0	99^0	99^8	100^0	98^6	99^8	100^0	100^2	100^0	100^0	99^4
NPN, mg. %	42	...	Operation	....	85	83	50	48	....	....	46	....	....	....	32
Urea clearance, %	62	...		....	....	....	20	....	....	....	....	....	....	....	50
Hippuric acid test, %	..	121		....	....	....	50	....	....	....	....	....	....	....	80
Prothrombin, %	91	...	....	....	66	60	....	....	....	....	89				
Icteric index, units	8	...	....	....	....	60	....	....	....	....	....	....	....	....	10
BSP test, % retention	0	...	....	....	20	....	....	....	....	....	....	....	....	0	
24 hr. fluid intake cc	..	...	3000	4500	4200	1200	4300	3500	3500	3800	2000	150	2250	2000	2250
24 hr. urine output cc.	..	...	500	340	90	210	1230	2250	2000	1170	940	1000	600	480	1260
Decholin, 20%, 10 cc.	..	...	....	1	1	1	1	1	1						
Parenteral glucose, Gm	..	...	200	200	300	400	250	250	250	50	50				

Tests of liver function were all normal before surgery (Table VII); hippuric acid test 121 per cent, prothrombin time 91 per cent, bromsulfalein test 0 retention in thirty minutes. Urea clearance (62 per cent) was slightly subnormal and a mild degree of nitrogenous retention was present on admission (NPN 42 mg. per cent, urea N. 24 mg. per cent).

Cholecystectomy under spinal anesthesia was uneventful except for a transient fall in blood pressure to 70/60 mm. Inhalation of 95 per cent oxygen—5 per cent carbon dioxide and 0.5 cc. adrenalin subcutaneously, promptly restored the pressure where it remained between 120 and 130 mm. The liver appeared grossly normal. The gallbladder was distended with clear bile and contained many variable sized stones.

During the first twenty-four hours after operation the course was not unusual except for diminished secretion of urine. (Table VII.) On the day following surgery continuous gastric drainage was begun because of nausea and frequent emesis. Despite a parenteral intake of One hundred Gm. of glucose with 10 cc. of decholin sodium were given daily for five days before operation.

4,500 cc. of fluids containing 200 Gm. of glucose the oliguria persisted. Thirty-six hours after operation the sclera had a definite icteric tint and the patient looked ill. The blood pressure was sustained at its original level.

Blood chemistry and function tests forty-eight hours after operation indicated appreciable impairment of both liver and kidney function. The NPN was 85 mg. per cent, there was 20 per cent retention of dye in thirty minutes with the bromsulfalein test and the prothrombin time had dropped from the preoperative level of 91 to 66 per cent. Clinically the patient looked haggard and complained of nausea and weakness. The abdomen was flat and the gastric drainage bile stained. The pulse rate was 120, quality poor and the blood pressure 92/70 mm. A mild cyanosis and icterus were present on the evening of the second postoperative day when oxygen therapy was instituted (6 liters per minute by nasal catheter).

During the second postoperative day 4,200 cc. of parenteral fluids, 300 Gm. of glucose and 10 cc. decholin sodium were administered. The twenty-four-hour urine output was 90 cc. The patient appeared critically ill.

On the third postoperative day the appearance was somewhat improved. Oxygen was discontinued. The blood pressure was 160/95, the pulse 116 and of good quality, the temperature 100.4°F. The blood NPN was 83 mg. per cent, urea N 60 mg. per cent, prothrombin 60 per cent, icteric index 60. One ampule of decholin sodium and hypertonic glucose (400 Gm. in 1,200 cc. of fluid) was given in small infusions throughout the day in the hope of breaking through the urinary suppression. The urinary output for the twenty-four hour period was 210 cc.

On the fourth postoperative day the patient was extremely restless and confused at intervals. The blood pressure was 154/86, the pulse 116 and regular, and the temperature 99.2°F. An intravenous hippuric acid test was 50 per cent of normal in contrast to 121 per cent preoperatively. Despite the oliguria the NPN had dropped to 50 mg. per cent. Forty-three hundred cc. of parenteral fluid containing 250 Gm. of glucose with 10 cc. decholin sodium resulted in a twenty-four-hour urine volume of 1,230 cc.

On the fifth and sixth days active diuresis was obtained on an intake of 3,500 cc. containing 250 Gm. of glucose and 10 cc. decholin sodium. (Table VII.) Gastric lavage was discontinued. From this point on the patient made an uneventful recovery. By the eighth postoperative day the prothrombin time had risen to 89 per cent without vitamin K therapy. On the thirteenth day a hippuric acid test was 80 per cent, there was no retention of dye in thirty minutes with the B.S.P. test, the skin had cleared of the jaundice and the blood NPN was normal.

Comment. This patient, as the others in group III, presented herself for surgery with kidneys having impaired reserve. The laboratory evidence of impaired hepatic function after surgery was more evident than in Case VIII, i.e., jaundice, lowered prothrombin, impaired removal of bromsulfalein dye as well as a lowered hippuric acid synthesis. However, impaired renal function invalidates the hippuric test in this instance. After better than sixty hours of pronounced urinary suppression a diuresis occurred following hypertonic glucose and decholin therapy. It is interesting and perhaps significant that the azotemia decreased in the presence of persistent oliguria. The fall in blood NPN and urea N preceded the increased urine output by a number of hours. It is to be noted that the blood NPN reached its highest level forty-eight hours after operation where it remained twenty-four hours despite a urine volume of 90 cc. for that period. Concomitant with the improvement in kidney function and clinical appearance the prothrombin value increased promptly without specific therapy, indicating rapid recovery of this particular hepatic function. The character of the temperature curve (Table VII) is suggestive of an associated mild infectious process of undetermined origin.

Case X. H. G., a female, age sixty-six, entered the hospital with a typical history of intermittent biliary colic of ten years' duration. At onset attacks occurred once or twice a year but during the past two years they had become progressively more frequent so that during the past six months they occurred at weekly or

more frequent intervals. Of late morphine by hypodermic had been given for distress. There had never been associated jaundice, fever or chills. The attacks occurred at any time of the day or night.

Inventory by systems was non-contributory. A pelvic abscess had been drained in 1922.

Cholecystectomy was done under spinal anesthesia. The gallbladder was thick-walled, adherent to the liver bed and contained thick white bile and many faceted stones. The liver appeared normal. Except for a transient fall in blood pressure to 80/60 mm. which promptly returned to normal after 0.5 cc. adrenalin by

TABLE VIII
CASE X. H. G.

Days Pre- and Postoperative	5	4	3	2	1	0	1	2	3	4	5	6	7	8
Temperature	98^6	98^8	98^8	98^6	98^8	98^6	98^6	99^2	99^8	99^0	98^8	98^8	98^6	98^6
NPN, mg. %	48	..	40	...	36	....	....	60	54	46	50	....	42	38
Urea clearance, %	60	..	...	...	...	....	....	25	....	....	....	....	....	46
Hippuric acid test, %	..	74	...	...	86	Opera-	....	30	....	....	....	....	....	58
BSP test, %	0	..	...	...	...	tion	....	20	....	....	....	....	....	0
Prothrombin, %	86	..	...	...	...	....	....	83						
24 hr. fluid intake, cc	..	..	...	...	...	2500	2600	3500	2000	1500	1500	1800	1750	2000
24 hr. urine output, cc	..	..	...	...	...	1100	380	400	740	900	600	950	720	630
Decholin, 20%, 10 cc	..	..	...	...	...	1	1	1	1	1	1			
Parenteral glucose, Gm	..	..	100	100	100	200	250	300	150	150	150			

There had been five term pregnancies with normal deliveries and menopause in 1929 had been uneventful.

Physical examination revealed a well preserved, well developed, moderately obese woman in no apparent distress and without jaundice. Physical findings were essentially negative except for moderate tenderness in the upper right quadrant. The blood pressure was 166/84 mm., pulse rate 92 and regular in rate and force.

X-ray after dye revealed a non-visualizing gallbladder.

The patient was kept free of distress on atropine, nitroglycerin and two decholin tablets three times a day. Three days before surgery infusions of 500 cc. of 20 per cent glucose with 1 Gm. of calcium gluconate was given daily.

Laboratory work-up (Table VIII) revealed a slightly elevated NPN and urea N that declined to normal after preparation for operation. The hippuric acid test was subnormal (74 per cent), the urea clearance 10 per cent below the lower limits of normal (60 per cent) and there was no retention of dye at thirty minutes with the bromsulfalein test. The prothrombin time was 86 per cent. Following preparation for operation the hippuric acid test was 86 per cent and the blood NPN was within normal range.

hyperdermic and inhalation of 95 per cent oxygen-5 per cent carbon diozide, the operation was well tolerated. Six hours after operation the blood pressure was 178/110, the pulse 116, regular and of good quality. During the first twenty-four-hour period following operation 2,500 cc. of fluid was given parenterally containing 200 Gm. of glucose and 10 cc. of decholin sodium. A twenty-four-hour urinary output of 1,000 cc. was considered adequate. The same therapy was repeated during the second twenty-four-hour period except 250 Gm. of glucose were given. Urinary suppression was eminent with a twenty-four-hour output of 380 cc. During the afternoon the patient complained of nausea and fatigue and after several emesis of foul, dark material continuous gastric lavage was instituted. The temperature was normal, the blood pressure 164/88, the pulse 108 and regular.

On the second postoperative day the patient appeared listless but was in no distress. Laboratory studies gave evidence of impaired function of both liver and kidneys: intravenous hippuric acid test 30 per cent, bromsulfalein retention 20 per cent at thirty minutes, prothrombin 83 per cent, urea clearance 25 per cent, NPN 60 mg. per cent, urea N 58 mg. per cent. Thirty-five hundred cc. of parenteral fluid containing 300 Gm. of glucose and 10 cc. of decholin sodium was accompanied by a mild diuresis, the

twenty-four-hour urinary output reaching 1,400 cc. On the evening of the second postoperative day auricular fibrillation developed. Blood pressure was 154/56 mm., apex rate 156, radial pulse rate 96. The gastric drainage contained bloody fluid.

The same line of therapy except for lowered fluid intake because of the cardiovascular status was continued (Table VIII) throughout the third, fourth and fifth postoperative days with progressive clinical improvement. The heart rate became regular fourteen hours after the onset of fibrillation and gastric drainage was discontinued on the evening of the third postoperative day. With the twenty-four-hour urinary output between 600 and 950 cc. the nitrogenous retention decreased progressively to normal values on the eighth postoperative day. At this time the hippuric acid test and urea clearance were still impaired but were well above the values obtained forty-eight hours after operation. The patient was dismissed on the fifteenth postoperative day.

Comment. Any resemblance to the hepatorenal syndrome in this patient following cholecystectomy seems primarily due to poor kidney function. The drop in hippuric acid excretion cannot be ascribed to liver insufficiency because of the associated decline in urea clearance. We have seen normal hippuric acid excretion in patients in whom urea clearance was decreased 40 to 50 per cent of normal; however, below these values impaired kidney function invalidates the hippuric acid test as a reliable criterion of liver function. The retention of bromsulfalein, however, is indicative of definite hepatic dysfunction, although the prothrombin value of 83 per cent is against any degree of markedly impaired liver function. It is noteworthy in this case that following the onset of auricular fibrillation which contraindicated large volumes of intravenous fluids, the nonprotein nitrogen dropped to normal values despite the fact that urinary output remained at levels which are supposedly inadequate to clear the blood of nitrogenous waste products in the presence of impaired kidney function. Decholin sodium therapy may possibly have influenced the existing azotemia by some unknown effect on urea and nitrogen metabolism.

COMMENTS

Vital organs, of which the liver and kidneys are examples, are characterized physiologically by large functional reserves—large factors of safety. It has been estimated that but one-fourth of the parenchyma of these organs functioning *normally* is sufficient to carry out bodily functions. Decompensation follows when functional reserves are exhausted. Laboratory tests fall into two categories: *function tests*, such as the hippuric acid and urea clearance tests, and *decompensation tests*, such as determinations of nitrogenous retention or the quantitation of icterus. The former give us insight to the state of functional reserve. The latter give an idea of severity of parenchymal damage in an organ whose functional reserve has been depleted. There is no justification for the assumption that liver and kidney function are "normal" in a patient who is not jaundiced and whose NPN is within normal limits. This situation obtains not infrequently in patients who are without functional reserves and who are thrown immediately into decompensation by insult that would ordinarily be considered insignificant. Yet much of our present knowledge of the hepatorenal syndrome springs from such a premise.

Considerable supportive evidence of a postoperative liver-kidney relationship is indirect. In many instances conclusions have been reached by *a priori* interpretation of hospital records and necropsy material. The functional state of the organs under indictment prior to the onset of pathologic decompensation was problematical or unknown. This is particularly true of kidney function. Bartlett[2] has called attention to this shortcoming. Before postoperative renal insufficiency can be attributed to extrinsic origin appropriate tests which demonstrate previously existing normal renal reserve must be on record.

There is a paucity of such data in hepatorenal literature.

Certain clinical and laboratory findings were so prevalent in our group of patients that they deserve evaluation as potential forerummers of the so-called hepatorenal picture in surgical patients.

Compensated Renal Insufficiency before Surgery. Nine of our ten subjects had a normal NPN before surgery. However, eight of the ten possessed varying degrees of diminished renal reserve which was revealed by subnormal urea clearance. Obviously, the development of a hepatorenal picture in these patients stemmed from kidneys with impoverished reserves.

Vasomotor Instability. Anoxia during or following surgery is an important predisposing factor in the hepatorenal picture. Seven of the ten patients in our series developed delayed postoperative hypotension. Five of the seven had previously been exposed to transient fall in blood pressure to shock levels while on the operating table. This followed, frequently but not always, traction on the mesentery or biliary tract. Only two of our subjects experienced a drop in blood pressure during the operation without a recurrence of hypotension during the postoperative period. This is significant. In all instances except Case VII the blood pressure was restored during the operation by subcutaneously administered adrenalin and 95 per cent oxygen-5 per cent carbon dioxide by mask. Four of the six patients developed hypotension, 70 mm. systolic or less, within twelve hours. In the remaining two, vasomotor collapse was delayed twenty-four and forty-eight hours, respectively.

We have observed significant drops in hepatic function in healthy surgical patients following transient shock during operation.[28] The poor risk patient with limited hepatic and renal reserves tolerates a like degree of anoxia even more poorly if at all.

Factors other than anoxia may be at work in patients who exhibit such a tendency to vasomotor collapse. Boyce[5] suggests that such reactions may be anaphylactoid in nature: following operation there is released by the liver a hypothetical toxic substance to which the patient has become sensitized. Anaphylactoid shock follows. Only indirect experimental evidence supports such a view. Based on more concrete experimental evidence is Ravdin's vasodepressor theory.[25] This investigator has extracted vasodepressor substances from the livers of dogs with obstructed common ducts. An autoclaved, alcoholic extract of fresh normal liver produced less of a drop in blood pressure (anesthetized cat) than a comparable dose of jaundiced liver extract. A dose of extract representing as little as 40 mg. of jaundiced liver substance produced a fall in pressure. Obstructed livers contained more histamine and choline than normal livers. Working along a similar line of investigation, Boyce[5] extracted the livers of patients whose postoperative clinical course and microscopic sections of liver and kidney conformed with the hepatorenal picture. Dogs injected with saline and watery extracts showed parenchymatous degeneration of the liver cells with comparable changes in the convoluted tubules of the kidneys. At autopsy a dog injected with alcoholic liver extract showed no significant findings. Neither blood pressure determinations or blood chemistry studies were reported in the latter's studies. Presumably the two investigators were dealing with different liver fractions inasmuch as Ravdin's vasodepressor substances were alcohol soluble while Boyce obtained positive results only with saline and aqueous pathologic liver extracts.

Early Hepatic Insufficiency, Urinary Suppression and Azotemia. Nine of ten patients gave evidence of decreased liver function within twenty-four to forty-eight hours following operation. However, the earliest sign of impending trouble was urinary suppression despite adequate fluid intake. As a rule, urinary suppression became most marked during the second twenty-four-hour postoperative period al-

though in half of the cases oliguria developed within the first twelve hours. Because of impaired renal clearance results of the postoperative hippuric acid test were considered invalid. Prothrombin time, dye elimination, icteric index or blood bilirubin levels, and in two instances, bile salt concentration in drainage bile were used as liver function criteria.

It is our impression that the secretory activity of the kidney is compromised earlier and to a greater degree than hepatic function during the same interval. In three subjects who recovered from what appeared clinically as a critical, fully developed hepatorenal picture (Cases IV, IX and X) the onset of diuresis was followed by clinical improvement. Curiously, patients IV and IX appeared to have passed the crisis and the blood NPN had begun to decrease *six hours before* active diuresis was an actuality. On the contrary, improved renal elimination in patients I, II, XVII and VIII was without effect on the fatal course. In the remaining three subjects oliguria and azotemia were progressive.

The ability of the liver to respond to specific therapy in one or more of its functions after the hepatorenal picture is well developed was surprising and is worthy of comment. Patients with subnormal hepatic function who respond poorly to specific therapeutic measures directed at improving liver function tolerate operative procedures much more poorly than patients who, although poor risks, show improved liver function after the same preparation.

CONCLUSIONS

1. The liver plays an important rôle in postoperative morbidity and mortality. Patients with evidence of subnormal liver function who responded poorly to specific measures directed at improving hepatic function were poorer surgical risks than those who responded favorably.

2. Patients who developed hepatic and renal insufficiency following surgery usually gave evidence of subnormal renal reserve prior to operation.

3. Diminished renal reserve plays a greater rôle in the development of the hepatorenal syndrome in surgical patients than is generally recognized. Knowledge of the blood NPN is without information on renal reserve until such reserves have been depleted.

4. Transitory fall of blood pressure and anoxia in poor risk surgical patients should be anticipated during and after surgery. Preventative treatment is the most effective treatment of circulatory collapse.

5. Decholin sodium appears to have produced favorable results in the treatment of postoperative hepatic and renal insufficiency.

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DOUBLE FRACTURES AND DOUBLE NON-UNIONS OF THE SHAFT OF THE TIBIA

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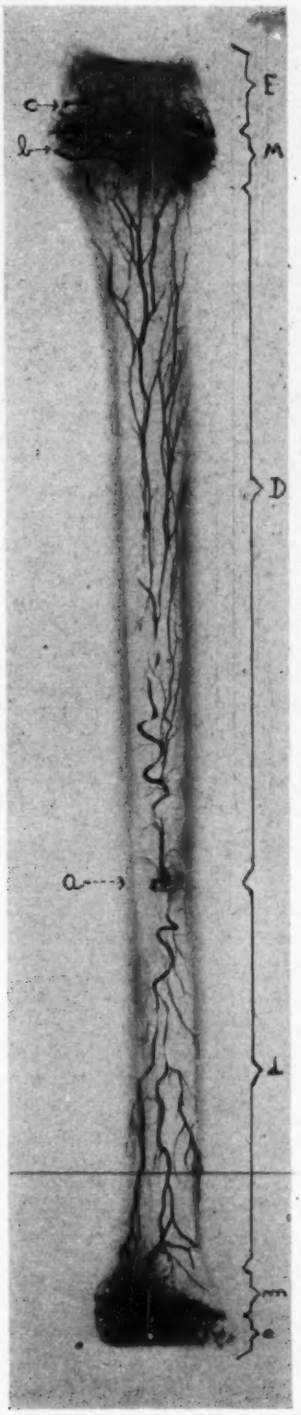
DOUBLE fractures of the shaft of the tibia are not infrequent. Their healing process is slow and the incidence of non-union high. The cause of it is the disturbance of the intra-osseous circulation following such fractures.

Anatomically, there are two vascular systems in the normal long medullated bone (Langer, Lexer, Delkes-Kamp and Kolodny,) the periosteal and the intra-osseous. The periosteal system consists of a net of smaller and middle-sized vessels which are derived directly from the surrounding soft tissue and are in intimate connection with them. Only a few small anastomoses connect the periosteal with the intra-osseous circulation. The intra-osseous system consists of three sets of vessels (Fig. 1): The first set is that of the diaphysis containing the nutrient artery which enters the bone as a single trunk except at the femur when it is double. (Fig. 1*a*.) Very soon it splits up into an ascending and descending branch and crosses toward the metaphysis. The second set is formed by the metaphyseal vessels (Fig. 1*b*) which are branches of the vessels of the joint capsule. They penetrate the bone near the insertion of the capsule beneath the epiphyseal line and supply the metaphysis. The third set is that of the epiphysis (Fig. 1*c*), the vessels of which are derived from the joint ligaments. In youth the three intra-osseous sets are connected only with a few anastomoses. With growth the anastomoses become more numerous, and after the period of development the three sets form one system.

From these considerations it becomes evident that the periosteal and intra-osseous system have only a few anastomoses. Hence the bone itself is chiefly vascularized by the intra-osseous system. In an ordinary single fracture of the shaft of the tibia, both fragments remain well vascularized even if the fracture should happen to be through the entrance of the nutrient artery, in other words, in level of the foramen nutricium. (Fig. 2*a*.) The collateral circulation from the metaphyseal vessels is sufficient to assure adequate blood supply to both fragments. In a double fracture of the shaft of the tibia, however, the middle fragment may become deprived of the bulk of its blood supply (Fig. 2*b*) unless it contains the nutrient artery.

As a rule, in the human tibia the nutrient artery enters the shaft within the proximal third at the posterior surface near the linea poplitea. Hence, in a double fracture in which the proximal fracture line runs through the foramen nutricium or distal to it, the middle fragment is apt to become deprived of its blood supply and may become necrotic. If this is the case, the incidence of non-union is high. The middle fragment can become regenerated only by ingrowth of vessels from the proximal and distal fragments and from the surrounding periosteum. This process is slow and non-union may develop in the meantime. In such a case the best treatment is complete and prolonged immobilization of the limb in a skin-tight plaster cast to promote and not to disturb the ingrowth of vessels from the peripheral fragments. If the proximal fracture line runs proximal to the foramen nutricium, the central fragment remains vascularized unless the nutrient artery becomes injured from the trauma and displacement of the fragment. If it remains vascularized, the healing



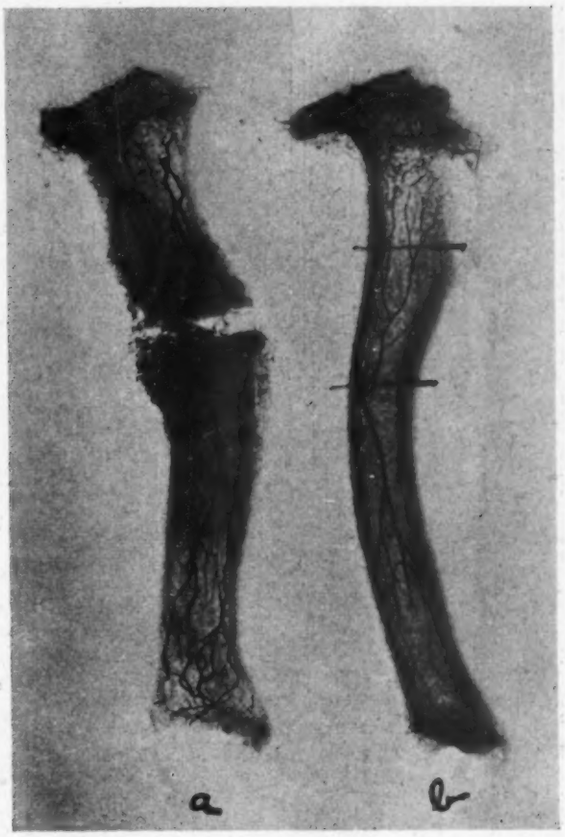
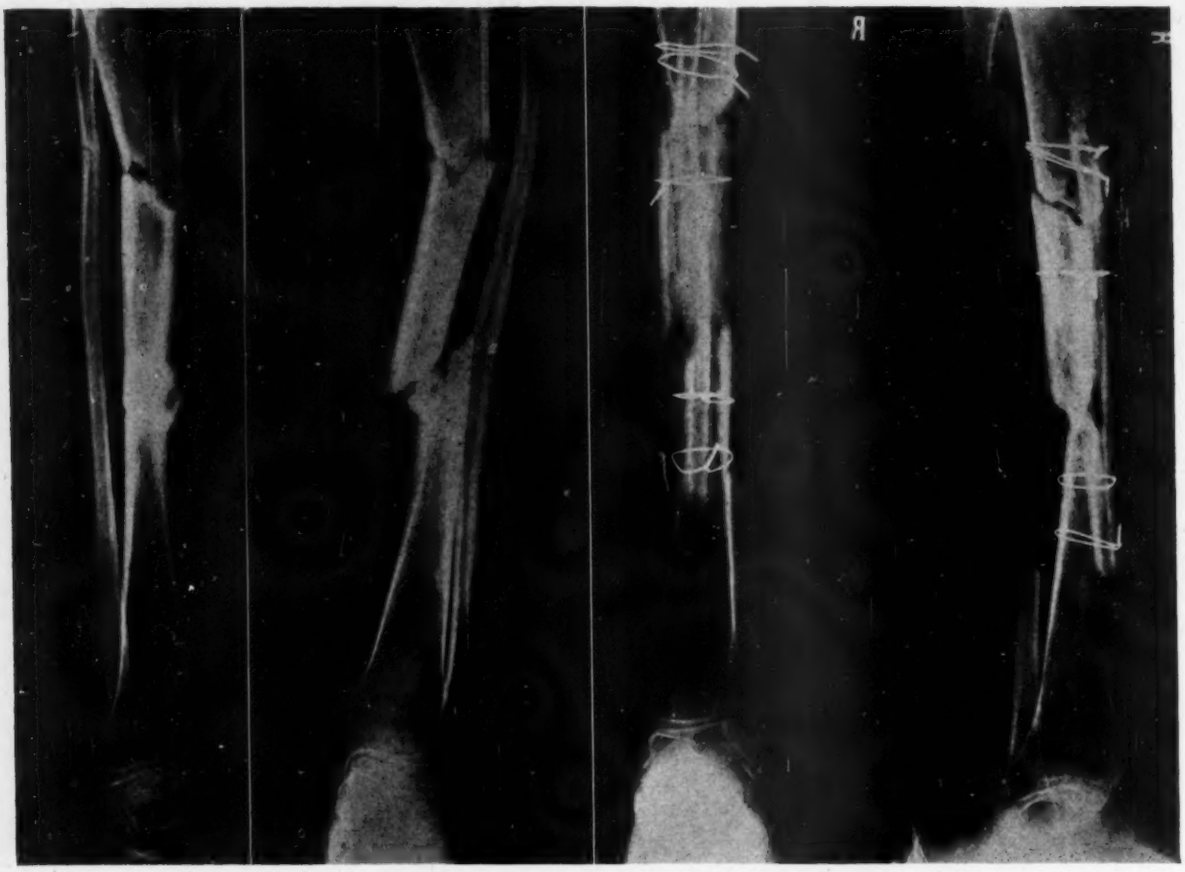


FIG. 2. *a*, Experimental fracture of the radius of a dog three weeks after the fracture. The fracture line runs just distal to the entrance of the nutrient artery. Both fragments are well vascularized, the proximal fragment from the descending branch of the nutrient artery and the distal fragment from the collateral circulation from metaphyseal vessels. *b*, Double fracture where the fracture lines would run through the entrance of the nutrient artery and the second one distal to it; the middle fragment would be deprived of the interosseous circulation.

FIG. 1. Representation of the bone vessels of the radius of a dog. The dog was killed under anesthesia. Immediately afterward the shoulder girdle was disarticulated and the vessels injected with a turpentine mercury solution through the arterial axillaris. The radius was removed. The periosteum was scraped from the bone and an x-ray picture was taken. The distal part (DME) is still in the stage of growth. Hence, the vascular systems are still separated from each other. The proximal part (*dme*) is beyond the period of development. The vessels have undergone anastomosis. *a*, Entrance of the nutrient artery; *b*, entrance of the metaphyseal artery; *c*, entrance of the epiphyseal artery.



A B

FIG. 3. A, double non-union of the tibia seven months after the accident. The middle fragment is apparently deprived of the interosseous circulation and has undergone aseptic necrosis as evidenced from the density of the fragment. (Compare with Fig. 2*b*.) B, the shaft of the fibula was transplanted to the tibia bridging both non-unions.

process of the fracture will take the ordinary course.

From roentgenologic studies alone it is difficult to judge whether the proximal fracture line runs above or below the entrance of the nutrient artery since only rarely would it be possible to visualize the foramen nutricium. Hence it is safer to treat a double fracture of the shaft of the tibia rather by complete immobilization than by traction.

Later studies, however, are of value concerning the changes of the bone structures. If the middle fragment does not atrophy in the same degree as the proximal and distal fragments do, in other words, if it remains dense, it must be assumed that it is deprived of its circulation which otherwise would permit the calcium to be carried away. (Fig. 3A.) If non-union has developed, obviously the best way to treat it is by bone grafting. The bone graft, however, should be long enough to bridge both non-unions. This would require a long massive graft from the tibia of the other side. This, however, may weaken the other leg. To avoid this the author advises the use of a fibula graft taken from the same side.

CASE REPORTS

CASE 1. A patient, aged twenty-one, sustained a fracture of the right fibula and a double fracture of the right tibia in a motorcycle accident. The fibula fracture healed; the double fracture of the tibia had not healed seven months after the accident. Note in the illustration the density of the middle fragment as compared with the atrophy of the proximal and distal fragments. Hence from x-ray examination it was evident that the middle fragment was dead. To promote healing of the non-

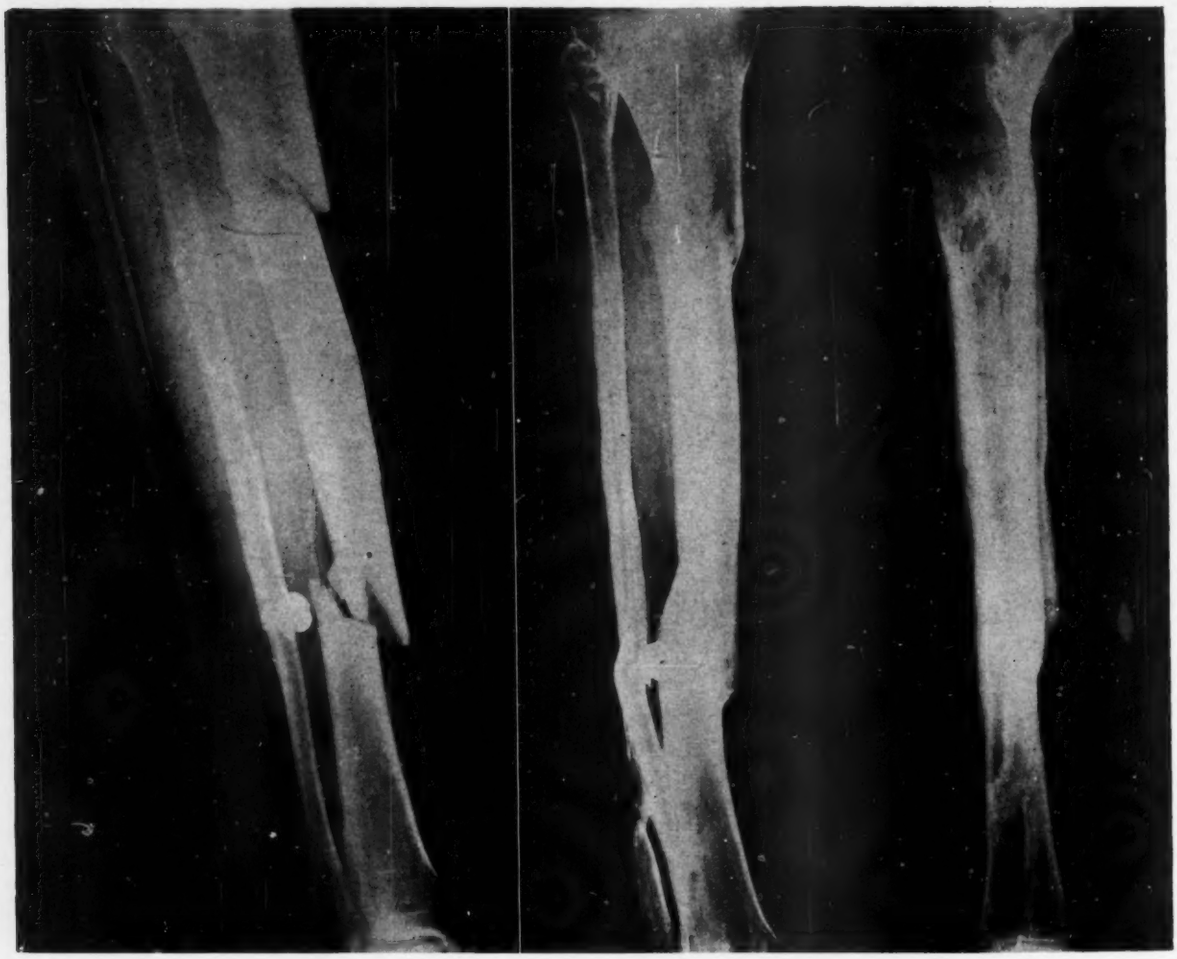
union bone graft operation was performed. The tibia was exposed from an anterior incision; the middle fragment was removed subperiosteally. It then was halved and the posterior half was replaced. The proximal and distal fragments were prepared for reception of the bone graft, i.e., the anterior cortex was removed. From a lateroposterior incision the fibula of the same side covered with its periosteum was removed. It was laid upon the three fragments and fastened with wires. An x-ray taken five months after the operation showed satisfactory healing conditions. The patient was discharged in a walking brace. About a year after the operation the non-union cleft became less visible and the middle fragment organized. In order not to delay regeneration the wires were removed. Two years after the operation, graft and host bones had become an organic union. The patient walked without support or a limp. (Figs 3A to C.)

Grafting of the fibula or of a large massive bone graft is an extensive operation. If the patient's general condition is such as to make this procedure unsafe, the writer recommends the transfer of the fibula to the tibia of the same side in stages. Hahn (1884) was the first to conceive the idea of fibular transfer. He was followed by Codivilla. The method was subsequently improved by Huntington, Stone, Campbell, Wilson and Myerding. The technic varies with the age of the patient. In adults the author prefers fusion of the upper tibiofibular joint in the first stage (Campbell, Meyerding), and an osteotomy of the lower part of the fibula and fusion of this part with the tibia (Wilson) in the second stage. The method has many advantages: the fibula does not become separated from its blood supply; the transferred and fused fibula provides a solid strut and it also permits immobilization of the tibia so that non-unions may heal even without subsequent bone grafting.

Case II. A patient, aged fifty-two, was struck by an automobile. He was admitted unconscious with fractures of the right clavicle, right radius, eighth rib, right fibula and double fracture of the right tibia. He regained consciousness soon. His general condition remained precarious. No treatment was required for the fractured clavicle; fracture of the radius was reduced and immobilized; a skin-tight plaster cast was applied to the right leg from his toes to the mid-thigh. All fractures healed with the exception of the double fracture of the tibia. The middle fragment became dense while the proximal and distal fragments underwent atrophy. These x-ray findings made it evident that the middle fragment was deprived of adequate circulation and had undergone aseptic necrosis. A major bone grafting operation did not appear advisable since the general condition of the patient remained impaired. Hence a fibula transfer in two stages was carried out four months after the accident. (Figs. 4A and B.) The first stage included fusion of the upper tibiofibular joint. From an incision parallel to the peroneal nerve the latter was located, dissected free and held away with a tape. The head of the fibula was now freed from its ligaments. The opposing surfaces of the tibiofibular joint were thoroughly denuded by removing cartilage

C

Fig. 3. C, two years after the operation graft and host bone have become an organic unit.



A B

FIG. 4. A, double non-union of the tibia four months after the accident. B, fistula transferred in two stages. First stage, fusion of upper tibiofibular joint (note bone peg through head of fibula into the tibia); second stage, six weeks later osteotomy between middle and distal third of fibula and fusion of proximal fragment of fibula to tibia. This roentgenogram was taken one year after the first operation.

and periosteum with chisel and curette. A hole was excavated in the tibia opposite to the head of the fibula until the median surface of the head of the fibula could be almost inserted into the tibia. To obtain better union a canal was drilled through the head of the fibula and tibia and a bone peg was inserted which was removed from the upper anterior crest of the tibia. I recommended this since it provides a firmer internal fixation and the autogenous bone graft promotes quicker and firmer fusion. The second stage was undertaken six weeks later. With a tourniquet applied the ankle joint was exposed from an anterior incision between the tibia and fibula. The underlying tendons and muscles were retracted on either side until tibia and fibula were clearly visible. The fibula was now osteotomized with a chisel about 8 cm. above the ankle in an oblique plane from below inward to above outward. The lateral surface of the tibia was now exposed. With a blunt instrument, a hole was made in the interosseous membrane and the muscles anterior to it. The shaft of the fibula was now pulled through this hole and approximated to the tibia. At the point of approximation a flap of tibia cortex (the base being distal and lateral) was split. A groove was excavated medial to it for reception of the fibula. The fibula was now inserted into the prepared tibia. During this manoever the fibula fractured. After the fracture occurred the transfer of this part of the fibula was much facilitated. After closure of the wound a plaster cast was applied from the toes to the mid-thigh for a period of one month. It then was removed and replaced by a cast in

which a walking iron was incorporated. This cast was removed after two months. Swelling of the leg from poor circulation (arteriosclerotic changes) subsided gradually upon physiotherapy. The patient walked on crutches.

Eight months after the last operation the patient fell and fractured the femur of the same side through the intertrochanteric line. Insertion of a Smith-Petersen nail was performed according to the Engel-May method with application of an intertrochanteric attachment. Seven days after this operation the patient fell out of his wheel chair upon his right leg. All fractures held. He was up and around on crutches thirteen days after the operation. He walked without any support ten months after the second operation and two months after the nailing operation. The non-unions healed.

SUMMARY

The cause of the slow healing and the high incidence of non-unions of double fractures of the shaft of the tibia is due to the disturbance of the interosseous circulation following such fractures. If a double non-union develops, transplantation of the fibula of the same side as the bone graft is recommended, or the transfer of the fibula of the same side to the tibia in stages. In the first stage the upper tibiofibular joint is fused. To achieve firmer and quicker fusion insertion of a bone peg is recommended. In the second stage an osteotomy in the lower part of the fibula is performed and this part of the fibula is fused with the tibia.

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EXTRAPERITONEAL CESAREAN SECTION AS A PROCEDURE OF CHOICE

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A QUESTION that still remains in the minds of many obstetricians today is whether a true extraperitoneal operation is needed. Reductions in the mortality and morbidity of the various types of cesarean section have been so decided in the past twenty years that some men still question the superiority of the low transperitoneal retrovesical cervical operation when compared with the classical incisions (high or low). This may be true in the selective group of sections, but when one has to deal with frankly or potentially infected cases and with those in whom previous attempts at delivery have failed, all obstetricians agree that neither operation is effective in preventing peritoneal contamination. Despite tremendous experience, judgment as to whether craniotomy, the Porro operation or the extraperitoneal operation is the method of choice becomes difficult.

Each of these procedures has its advocates, and each advocate, I believe, is sincere in his conviction that he is doing what he thinks is right. Greenhill states, "In my experience the Porro operation is the safest one in the presence of definite infection." C. Buscoe finds, "the supposed protection given the infected patient by the Porro operation has been lacking in Philadelphia (1932–1944). Reports on extraperitoneal cesarean sections show the death rate from all cases to be from two to five times less than that from sepsis alone in cases in which the Porro operation was done." Monahan, Connally and Eastman believe, "that cervical cesarean section becomes increasingly hazardous after 18 hours of labor and that such cases had best be superseded either by cesarean hysterectomy or an extraperitoneal operation."

My experience with the Porro operation in frankly infected cases has been rather limited; but when I have had to resort to this operation, I have always removed the cervix as well and drained the pelvis. With the cervix allowed to remain, I can see no reason to defend the Porro operation in infected cases. Many series of cesarean hysterectomy can be cited with excellent results, but I question whether the showing would be any too impressive if these series were limited to infected cases only. Most of the Porro operations have been done for ruptured uteri, Couvelaire uteri, fibromyomas of the uterus, etc., in which intrauterine infections were absent.

Norton, Waters, Steele, Burns, Ricci and others prefer the extraperitoneal operation on the grounds that uteri are not removed needlessly and that babies are not sacrificed. Where there is probable or actual existence of intrauterine infection, they recommend the extraperitoneal operation. They claim that if the operation is properly performed postoperative peritonitis can be eliminated as a cause of death and that the uterus is conserved as well. To utilize this type of cesarean section in the very worst cases means that it must have virtues not obtainable in the transperitoneal operations. Some men (Henkel et al.) claim that the only feature in favor of the extraperitoneal section was that it prevented subsequent intra-abdominal adhesions. Sellheim and Kronig and in later years Beck and others believe that "careful walling off of the abdominal cavity with gauze was sufficient protection against infection."

Today we can truly dispute these statements. While the use of blood transfusions, sulfonamides, penicillin, streptomycin, etc.,

have made the lower segment two-flap type of cesarean section safer in those patients who have been in labor and those in whom the membranes have been ruptured for a period exceeding twenty-four hours, no conscientious obstetrician of today will admit that the intraperitoneal operation is as safe as the extraperitoneal types. The results to date with the extraperitoneal operations have been excellent but they do not tell the profession that it is a superior type of section. To do this one must be able to see what this operation can accomplish in large series of selective cases as well. Dr. H. Halsted, in discussing Waters's presentation of the "supravesical extraperitoneal cesarean section" before the New York Obstetrical Society in January, 1939, stated, "I feel with Dr. Burns that the extraperitoneal operation is an extremely valuable operation, that it is not done often enough, and I would like to see it developed, so that it can be used on patients who have not been in labor." Five years before this (1934) G. Kaboth reported on 308 extraperitoneal sections and considered it the least trying type of cesarean section for the mother and recommended it for all cases requiring delivery by abdominal section. Stimulated by Kaboth's report and Halsted's suggestion, the writer has performed the extraperitoneal operation as his routine method of section in elective cases to convince himself that the following statements were correct:

First. Any obstetrician with sound surgical background and training can overcome the technical difficulties entailed in performing the extraperitoneal operation. Stearns, after performing the Waters's supravesical operation in sixteen infected cases, opened the bladder inadvertently in three cases and invaded the peritoneal cavity in seven cases. He concluded, "This procedure should definitely relegate the Porro operation to its proper place in oblivion, save an occasional case." In my humble opinion, 43 per cent peritoneal perforations and 18.7 per cent bladder injuries do not spell success in surgery even though no mothers were lost, nor does it help to advance the cause for extraperitoneal cesarean section. With such results, it might be wiser to relegate the extraperitoneal operation to its place in oblivion and resurrect the Porro operation. Stearns further stated, "Extraperitoneal cesarean section need not and should not supersede low cervical intraperitoneal cesarean section, for the technique is learned with some difficulty and is sufficiently complicated that the average obstetrician needs a certain amount of training to become proficient. There should be in every large community a few men trained in the procedure to salvage the occasional mother or child who cannot be delivered safely in any other way." Daichman and Pomerance reported their results with the Waters's supravesical type of operation in one hundred cases with no maternal deaths. Unfortunately again for the reputation of this excellent operation, they reported that in thirty-six cases, one or more openings in the peritoneum occurred; that, in their series seventeen bladders were injured, and that in one case vesicovaginal and vesico-abdominal fistulas occurred.

With such a high incidence of bladder and peritoneal injuries, it is not surprising that many obstetricians do not care to learn the extraperitoneal operations. While it is true that the extraperitoneal operation does carry certain real and vexing dangers to the peritoneum, bladder, ureters, and large vessels in the vesico-uterine space and broad ligaments, and that the occurrence of vesicovaginal fistulas is lamentable, nevertheless it has been the experience of many men now well acquainted with the extraperitoneal operation that these accidents were commoner in the earlier days of their experience and that later on these pitfalls were almost always avoided.

The author has had ample opportunity to try several extraperitoneal approaches to the retrovesical, infraperitoneal space and is convinced that the paravesical approach utilized, developed and advocated

by J. F. Norton has much to offer over the supravesical approach of Waters. It is less time consuming, is less apt to produce peritoneal or bladder perforations, and can be mastered by any well trained obstetrician. Regardless of which method is chosen, success or failure will depend upon a good knowledge of the fascial structures and their relationship to the lower uterine segment, the peritoneum and the bladder. The hazy early anatomical descriptions of the anatomists have been replaced by detailed descriptions of both the anatomy and the surgical technics by such present day contributors as Burns, Steele, Ricci, Marr, Waters, Norton, Cartwright, Furniss and others.

In 1920 Lichenstein noted "that the longer the patient was in labor, the less likelihood there was of injuring the peritoneum. This was found to be true only in cases in which the membranes had not ruptured. If the membranes had ruptured early, the os remained practically unopened and the cervix did not retract." He concluded, "that in these cases, the extraperitoneal operation is difficult." While the effacement and dilation of the cervix unquestionably cause the level at which the visceral peritoneum fuses with the uterus (i.e., the upper limit of the isthmus) to rise and thus facilitate the performance of the extraperitoneal operation, the writer does not believe that this factor makes the extraperitoneal operation technically more difficult for the skillful obstetrician; nor does his experience allow him to concede that the operation will be fraught with serious dangers to the ureters and bladder, or that the peritoneum will be unavoidably injured in many instances. Having performed extraperitoneal hysterotomy at four months and, in not a few instances, having performed the extraperitoneal operation in placenta previa in cases between six and eight months, the writer has come to the conclusion that this operation can be used freely as the procedure of choice in nearly all cases requiring cesarean section. In the writer's series of one hundred cases of extraperitoneal cesarean section, there were no injuries to the bladder, ureters or lateral pelvic veins. The peritoneum was button-holed in only three cases, an incidence of 3 per cent, and these were infinitesmally small. They were repaired in the manner recommended by Ricci and others by tenting the peritoneum. When one considers that in at least 20 per cent of the writer's cases previous low flap cervical sections had been performed, one must concede that a 3 per cent incidence of peritoneal perforations is extremely small. Furthermore, these three perforations occurred in the first fifteen cases of this series. In the last eighty-five cases no peritoneal rents were made.

It has been brought out by Norton, Waters and others that there may be limitations of the exposed extraperitoneal space in "deep pelves and fat women." The peritoneum in these obese patients is apt to be exceedingly thin and easily torn. With proper care, neither the deepness of the pelvis nor the thinness of the peritoneum should act as deterrents; rather they should caution us to greater delicateness in the handling of tissues. The presence of many basal retrovesical varicosities of considerable size can be seen, but these need not alarm the operator as the incision can frequently be placed so that these can be avoided. These can best be avoided by elevating the upper fascioperitoneal flap a little higher than usual. Those that must be incised can be easily controlled by Allis clamps. A word of warning at this point may not be out of place: Regardless of which approach is chosen, the operator should try to keep all of the dissection lateral to the lateral umbilical ligaments. He must not traumatize the area lateral to these ligaments or else some of the larger vesical veins or lateral pelvic veins will be injured. Bleeding here, while not alarming, can be troublesome. Performed with due consideration of these details, the operation can be kept almost bloodless.

Second. The operation is a safe one from the standpoint of morbidity and mortality. Shock is reduced to a minimum due to the fact that the intestines do not come in contact with the operative field. Since no intra-abdominal manipulations are possible, postoperative distention and ileus are practically unknown. There should, therefore, be no postoperative adhesions. While postoperative adhesions are quite rare in the cervical two-flap type cesarean section, they do occur and act as a potential threat to the patient for the rest of her life. In the writer's series of one hundred cases shock never occurred. In no case was a transfusion necessary. The amount of blood lost never exceeded that seen in a normal delivery. There was not a single case of ileus or postoperative abdominal distention. (Table 1.)

TABLE 1

ANALYSIS OF ONE HUNDRED CASES OF EXTRAPERITONEAL CESAREAN SECTION

Maternal Mortality	0
Maternal Morbidity (usual standards) (average)	0.5 days
No. Cases	
69	no morbidity
23	one day morbidity
6	two days of morbidity
2	morbidity of three days or more (both due to wound infections)
No. of babies born alive	100
Fetal deaths	6
1. Placenta previa—7½ months	
2. Placenta previa—6½ months	
3. Placenta previa—7 months	
4. Severe pre-eclampsia—7 months	
5. Congenital defects—multiple	
6. Mongoloid idiot	
All Born Alive but Died within 72 Hours	
Operating time average	30 min.
Wound infections	2
Days in hospital	9.12 days
No. drained	3
No. in whom sulfonamides were used	3
Peritoneal perforations	3
Bladder injuries	0
Ureteral injuries	0
Injuries to lateral pelvic veins	0
Distention	0
Difficulty in the delivery of babies	0
Urinary complications	0
Number of transfusions	0
Amount of bleeding	Normal
Thrombophlebitis and phlebothrombosis	0

Third. The delivery of a full term baby of normal or larger than normal size should be easily accomplished. In most of the reported series of extraperitoneal cesarean sections, delivery of the fetal head was accomplished by forceps or by a vectis or single blade. It is the writer's guess that in not a few cases this was responsible for injury to the peritoneum and bladder. With this in mind, the writer has developed a technic which, if followed, will render the delivery of the baby infinitely easier.

Version and breech extraction is performed routinely on all vertex cases and is practiced as follows: If the fetal back is located on the mother's left, the operator inserts his left hand through the cervical incision. The baby's forehead and face are grasped by the left hand and pushed in the direction of its chest to obtain the maximum flexion of the neck. The head is carried to the left iliac fossa and allowed to remain there. The external hand (right) then pushes the breech downward and to the right so that the feet can be grasped by the left (internal) hand of the operator. It is preferable to grasp both feet and this should always be possible. If only one foot is grasped, the posterior foot should be chosen. (If the anterior foot is grasped, the posterior leg may splint itself against the maternal spine during the extraction of the anterior leg.) Once delivered, the leg or legs are grasped by an assistant and extracted by traction toward the ceiling. Before the assistant exerts any traction the operator reinserts his hand (the right hand is now preferable), and gently pushes the head up into the fundus while the assistant pulls on the legs. Both movements should be synchronized. The legs are rotated and extracted so that their extensor surfaces face the mother's head. This allows the abdomen to present anteriorly (not back anterior as is the practice of many men in the delivery of breech cases through the vagina). Traction is now made upward toward the ceiling and the body of the baby is slightly rotated counter clockwise (45 degrees) until the anterior (left)

scapula of the baby is seen. In this position the baby's back is toward the mother's left and the baby's left or anterior scapula is presenting. The fetal legs and body are swung in the direction of the mother's right knee. This allows the left or anterior arm to be delivered with ease. To deliver the posterior arm, the fetal legs and body are now swung upward in the direction of the mother's left shoulder. This allows the right (posterior) arm to be delivered. Should the baby's head and back lie to the mother's right side, the same procedures are carried out only in opposite order: The delivery of the aftercoming head will be no problem if the body and head are now rotated through an arc of 135 degrees so that the baby's occiput comes anterior. With a finger in the baby's mouth, flexion is accomplished and maintained. Traction on the aftercoming head and body is not downward but upward toward the mother's head. This can be facilitated by downward pressure by an assistant's hand placed over the fundus of the uterus. Forceps to the aftercoming head have never been necessary. In not a single case of this series has the writer experienced the least difficulty in the extraction of the babies. When the above maneuvers are carried out, the ease with which the babies can be delivered is surprising.*

Fourth. The operation must not be too time consuming. While the success of most operations should not be measured by time, I believe that in cesarean section the shorter the time, the less the bleeding and shock and the smoother the convalescence. It was argued that the extraperitoneal operation was too time consuming to be practical. In the writer's cases the shortest time was eighteen minutes, the longest time forty minutes; the average time was thirty-five minutes when the supravesical approach was used and thirty minutes when the paravesical approach was used. The writer is now determined in his decision to continue the paravesical approach in all of his future cases.

Fifth. The operation can be performed with an empty bladder. Most operators distend the bladder at the beginning of the operation, empty it when the proper cleavage planes are found and refill it at the completion of the operation to test its integrity. It has been the writer's belief that this consumes time, exposes the bladder to increased risk of injury and makes dissection more difficult. In none of the writer's cases has the bladder been filled and in none of his cases has the bladder been injured.

Sixth. The operation should be possible as a repeat procedure in cases in which a test of labor is given in a pregnancy that follows a previous cesarean section. If the test of labor should prove a failure, an extraperitoneal cesarean section should still be possible. With this in mind, the writer has purposely resorted to an extraperitoneal operation in cases that had previous intraperitoneal two-flap cervical cesarean sections. It has been his finding that while the bladder is more firmly anchored to the lower uterine segment than normally, dissection was still possible without injuring either the peritoneum or the bladder.

CONCLUSIONS

1. After putting the extraperitoneal operation through a rigid test in one hundred cases, the writer has come to the conclusion that if the extraperitoneal operation is the safest operation for infected cases, it should be the safest in clean cases. The writer agrees completely with Kaboth that, "it is the least trying type of cesarean section for the mother," and, "that it can be recommended for all cases requiring abdominal section."

2. The dangers of postoperative hernia, even where drainage of the operative area is used, is reduced to a minimum.

* When spinal anesthesia is used and the uterus is firmly contracted, version and breech extraction may be a little more difficult; but it is still possible and is easier and safer to accomplish than extraction with forceps.

3. The operation can and should be performed with an empty bladder.

4. The babies can be easily delivered by version and breech extraction. The use of forceps should be avoided.

5. There should be no postoperative adhesions or ileus.

6. Shock is reduced to a minimum since no intra-abdominal manipulations are necessary and no bowel comes in contact with the operative field.

7. Hemorrhage should be minimal.

8. There should be no danger of postoperative peritonitis.

9. There should be ease of performance which increases as experience is gained.

10. It can readily be used as a routine procedure in place of the intraperitoneal operation.

11. It can be used after previous sections without greater difficulty.

12. It is not too time consuming; the average time should be thirty to thirty-five minutes.

13. Postoperative convalescence is smoother, morbidity is less and the hospital stay is reduced.

14. Bladder complications are absent.

When removal of the uterus is indicated, as for fibroids and other non-malignant conditions, E. W. Munnell, Meigs and many other gynecologists believe that surgeons and gynecologists should be qualified to perform total hysterectomies. He admits that this process is followed by a slightly higher operative mortality and one must be most careful not to injure the ureters. Nevertheless, in skilled hands he believes hysterectomy should be performed to avoid possible malignancy of the remaining cervix at some later date. Also, the female's sexual response is unaffected by removal of the cervix. In fact, removal of the ovaries need not affect libido. For all these reasons it is urged that all future operators become skilled in performing total, rather than subtotal, hysterectomies. (*Richard A. Leonardo*, M. D.)

MODERN MANAGEMENT OF MEGACOLON*

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OUR purpose in this paper is to present a concise survey of the various methods of treating megacolon which are employed today, the results obtained from each and the complications encountered. Pathology, diagnosis and etiology will be discussed in so far as they affect treatment. We believe that a study which presents the best and latest thought on the subject should prove of value to the general practitioner, the pediatrician and the surgeon who may be called upon to treat this condition.

In view of the fact that so-called congenital megacolon is encountered only once in every 8,000[28] or 9,000[5] hospital admissions or once in 10,000 autopsies,[7] it is not surprising that we are today little closer to an explanation of this phenomenon than was Finney[8] when he wote in 1908, "It is highly probable that more than one etiological factor is concerned in its production, as no single cause that has thus far been discovered will satisfactorily explain every case." His paper still stands as a classic and is well worth the attention of anyone interested in the subject.

Congenital idiopathic megacolon is rarely seen in an adult and for that reason the following case is reported:

CASE REPORT

A. M., No. 145023, a white woman, aged fifty-three, was admitted to the New York City Hospital on June 28, 1946, complaining of severe abdominal distention of forty-eight hours' duration. For as long as she could remember this patient had been subject to attacks of marked constipation and more or less abdominal distention. During the past ten or fifteen years she had been in the habit of taking two alophen pills twice daily for several days each week to relieve constipation and gaseous distention. Two years ago she suffered an acute attack of obstipation, abdominal pain and great distention. She had to take two whole bottles of magnesium citrate before she could secure relief at that time. There was no history of diarrhea, bloody or tarry stools. Her appetite, which was usually good, had fallen off lately and she had lost a considerable but unknown amount of weight.

The present attack resembled the others in that its onset was insidious, she could not evacuate her bowels and she observed a progressive enlargement of her abdomen. But her symptoms were more acute this time, with marked nausea, anorexia and dyspnea. She became frightened and for the first time sought medical advice.

Except as mentioned, her past medical history was negative. She had always been in good health and able to perform her duties as housewife and mother.

Physical examination on admission disclosed an acutely ill, poorly nourished and very apprehensive white female of fifty-three. Her temperature was 98.6°F., pulse 100, respiration 24 and blood pressure 150/90. The abdomen was markedly distended and tympanitic. Gentle palpation revealed no masses, spasm or tenderness, although the slightest pressure increased her general discomfort. The liver and spleen could not be felt. Rectal examination revealed a normal sphincter, an empty ampulla and no local obstruction. The remainder of the examination was negative. The blood count and urinalysis on admission were normal. A flat x-ray of the abdomen showed tremendous dilatation of the whole colon with gas and feces from the cecum to the lower sigmoid region where the distention abruptly ceased.

Efforts were made to decompress the bowel by intubation and to restore the fluid-electrolyte balance with infusions. The distention,

* From the New York City Hospital, Department of Surgery (Service of Preston A. Wade, M.D.) and Department of Pathology (James R. Lisa, M.D., Director).

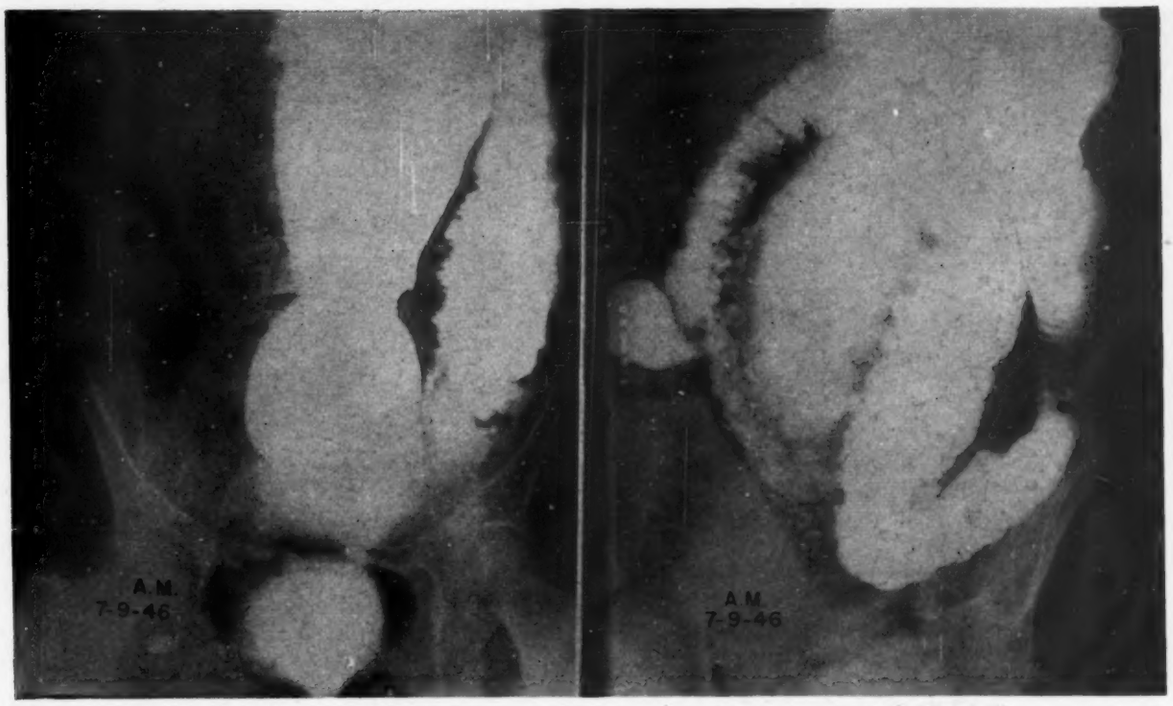

Fig. 1. Dilated, redundant sigmoid, ballooned out of pelvis, with obstruction due to volvulus at rectosigmoid.

however, was unrelieved. Further investigation of the distal colon by sigmoidoscopy and barium enema was considered too hazardous an undertaking in this acutely ill patient. Instead, with the patient prepared for operation, a spinal anesthetic was administered about twelve hours after admission. As no relief was afforded by this therapeutic test, an exploratory laparotomy was performed forthwith through a lower left paramedian incision for intestinal obstruction. The whole colon was tremendously distended with gas as far as the rectosigmoid. Distention was most marked in the descending colon and sigmoid which were enlarged to four times normal size. In this region the bowel wall was paper thin. The distal small intestine was dilated to a lesser degree. The descending and sigmoid mesocolon was extremely long and redundant. Due to the resultant abnormal mobility of this portion of the colon, an obstructing volvulus had occurred at the rectosigmoid junction. Manipulation of the bowel with reduction of the volvulus resulted in the sudden passage of a copious liquid stool with much gas. Marked immediate relief of the distention was observed. In view of the patient's poor condition no further surgery was attempted and the abdomen was closed.

The patient did well for a time after operation but the distention and obstipation recurred and persisted despite paravertebral blocks and spinal anesthesia. Her course for several weeks was marked with a septic type of temperature in spite of supportive measures which included infusions, transfusions and intubation. A barium enema (Fig. 1) confirmed recurrence of obstruction at the rectosigmoid. On August 8, 1946, under continuous spinal anesthesia (which incidentally, did not relieve the distention) 40 cm. of dilated atonic descending colon and sigmoid were resected by the Paul-Mikulicz method. The proximal clamp was removed at seventy-two hours but when the distal clamp dropped off at ten days it was found that the distal loop had retracted and closed over to such an extent that the spur could not be isolated.

The patient was therefore returned to the operating room, the distal loop was freed up and clamps were applied to the spur. In ten days these cut through and the patient began passing small stools per rectum. Her general condition meanwhile showed marked improvement. As the colostomy showed no signs of closing at the end of six weeks, operative closure was effected on October 15, 1946. The postoperative course was uneventful. On the fifth day normal bowel function was reestablished and the patient was discharged, free of symptoms, twelve days later.

FIG. 2. Hemorrhagic ulcerated mucosa of resected sigmoid.

The report of the pathologist (James R. Lisa, M.D.) was as follows:

"*Specimen No.* 29447—*Gross Appearance:* The sigmoid measures 40 cm. in length and 14 cm. in circumference. It has its mesentery attached. The serosa is discolored, hemorrhagic, cyanotic and granular, especially along the antimesenteric border. When opened, the specimen is found to be filled with blood. The wall is thick and oedematous and the inner surface is studded with innumerable petechial hemorrhages. In only limited areas is the mucosa normal. (Fig. 2.)

"*Microscopy:* The wall is greatly thickened and oedematous. It has a myxomatous appearance and is diffusely infiltrated with lymphoid and plasma cells. Beneath the serosa are groups of foreign body giant cells, forming granulomata. The vessels are engorged and there are numerous fresh hemorrhages in the subserosa. The mucosa is extensively eroded and its surface is covered with well vascularized granulation tissue. Ganglion cells of the plexus of Auerbach are present and appear normal in both the involved and the uninvolved portions of colon (Fig. 3), although in the involved area there is oedema of the plexus and infiltration with eosinophiles and polymorphonuclears."

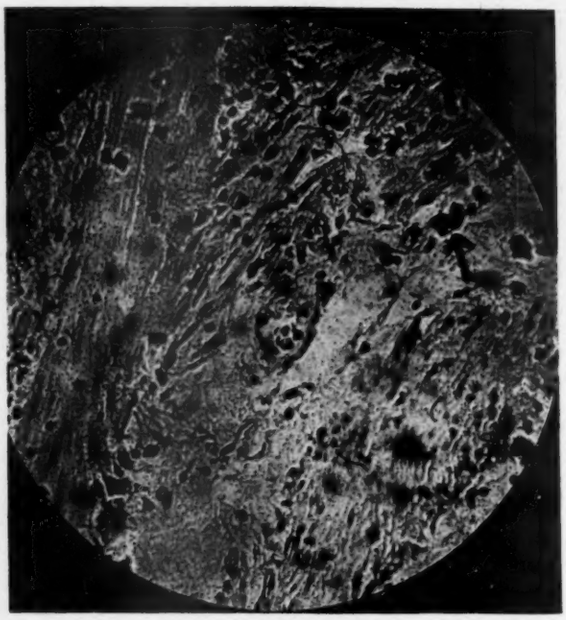

FIG. 3. Normal ganglion cells of Auerbach's plexus in wall of resected sigmoid.

On January 28, 1947, six months after admission to the hospital, the patient was seen at follow-up examination. She stated that she had never felt so well in her life, that she had gained 10 pounds and was having three or four soft-formed stools daily. She was taking half an ounce of mineral oil daily but had taken no cathartics or enemas. There was no evidence of any recurrence on physical examination. The abdomen was flat and soft with a well healed left rectus scar and no hernia. Rectal examination was negative. A barium enema the same day showed no evidence of obstruction or recurrence in the sigmoid but there was persisting redundance and enlargement of the remaining large intestine. (Fig. 4.)

PATHOLOGY

Before attempting to discuss the etiologic factors of megacolon it will be useful to consider briefly the basic pathologic anatomy involved. As its name implies, this is characteristically a disease of the colon. The small intestines are seldom implicated. Very rarely a portion of the terminal ileum may share in an extension of massive colon dilatation but that is all. According to many investigators distention of the rectum is unusual, but Grim-

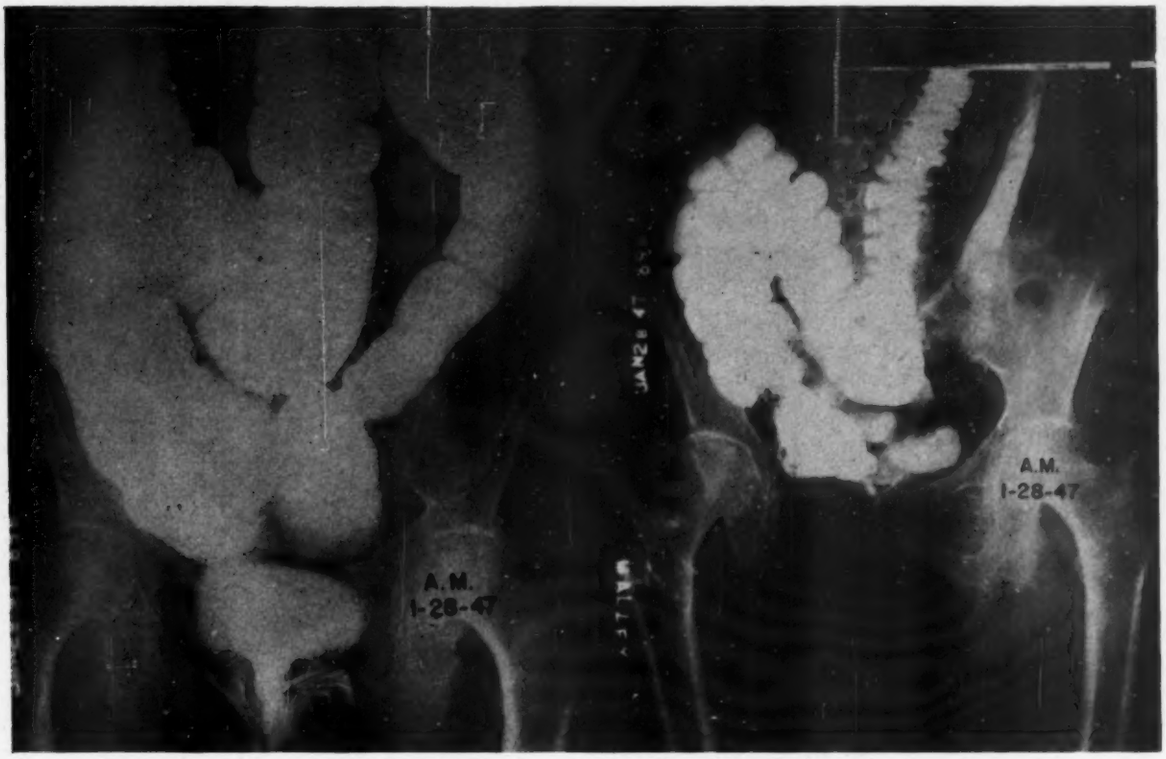

Fig. 4. Six months after operation; essentially normal sigmoid and descending colon; persistence of some redundance and enlargement of the remaining colon.

son[10] found it in half of his twenty-four cases. The whole large bowel from cecum to rectum may be involved (15 per cent of cases—Finney) but the sigmoid is the most frequent seat of the disease, being included in all or practically all cases.[8,10,18,21,24,33AA] Pathologic changes are limited to the sigmoid exclusively in more than one-third of cases.[2,8]

Dilatation is usually great and often tremendous. Bowel capacities of 40 pounds,[9] 16 liters,[23] 5 liters[30] and 4 gallons[27] have been reported. Hawkins[11] recorded a maximum circumference of 120.49 cm. and in Bach's case[1] the distended sigmoid measured 82.4 cm. in circumference and 10.5 inches in diameter. The transition from normal to dilated colon is usually gradual while that from dilated to normal bowel is most frequently abrupt. The lumen is sometimes distended with quantities of putty-like material having a sickeningly offensive odor, plus variable amounts of liquid feces and gas. Not infrequently large, stony-hard fecal concretions, sometimes as large as a child's head,[30] have been found impacted in the bowel.

The colon wall in a typical case is greatly hypertrophied, being several times its usual thickness, and it has aptly been compared with a wet leather glove in appearance.[33] On the other hand an occasional case, such as ours, at operation shows an attenuated paper-thin colon, stretched to the bursting point,[4,5] in place of the usual hypertrophy. Even in such cases, however, the fixed specimen may show a thick apparently hypertrophied wall. When hypertrophy is present, it is seen in all layers including the mucosa which is pigmented and often ulcerated.

In practically every case the diseased colon is elongated and redundant as well as hypertrophied. The mesocolon also partakes of this abnormality. It is much enlarged, thickened and gorged with swollen blood vessels, lymphatics and nodes. Rarely is it shortened.[18]

Histopathology confirms the changes seen in the gross specimen. With few ex-

ceptions there is extreme hypertrophy of all the colon layers and nothing else. Rarely, as in the present case, thickening of the wall proves to be pseudohypertrophy, a myxomatous condition produced by edema and round cell infiltration. Sometimes the mucosal surface is ulcerated. Few reports have been found in which examination was directed specifically to the plexuses of Auerbach and Meissner. Finney[8] stated that these ganglia were examined and found normal in two cases while Tiffin[32] reported complete absence of Auerbach's ganglion cells in 5 to 7 cm. of normal upper sigmoid just distal to a grossly dilated colon in a twenty-one-month male child. In our case the plexus of Auerbach was normal in the uninvolved portion of the resected colon; in the diseased areas, however, it was edematous and infiltrated with polymorphonuclears and eosinophiles but the ganglion cells were apparently normal. (Fig. 3.) It is unfortunate that this investigation has been carried out so seldom in the past as it might throw some light on the etiology of the disease.

DIAGNOSIS

The clinical picture is essentially one of obstipation and meteorism. Although seen most frequently in the very young (70 per cent in the first few weeks of life—Finney) it may become manifest at any age. It is well established that megacolon in some cases is compatible with comparatively good health and may go unrecognized for several decades. It occurs predominantly in males.[5] Typically there has been severe constipation either from birth or over a long period of years. There may on occasion be alternate episodes of diarrhea which give no relief and fail to empty the bowel. Spontaneous stools are the exception. Weeks may pass without an evacuation and undigested food may then be noticed in the stool. There is progressive abdominal enlargement which remains unrelieved by the infrequent stools. Weight loss is progressive and may be extreme. Not infrequently the outline of the distended colon can be traced through the thinned-out abdominal wall and vigorous peristaltic contractions can be seen. Whitehouse[33] mentions the doughy feel of the abdomen and the putty-like fecal masses which can occasionally be palpated. Respiratory embarrassment from pressure of the enormously distended gut is not uncommon.

ETIOLOGY

Hirschsprung[13] divided megacolon cases into groups according to age: those occurring in infancy which he called true megacolon, and those occurring in adult life which he considered pseudomegacolon. Age, however, is not a reliable criterion, for colonic dilatation due to organic obstruction occurs in infants and also megacolon which is, so far as we can tell, congenital and idiopathic, occasionally presents itself in an adult.

Enlargement and hypertrophy of the large bowel, at any age, secondary to recognizable organic obstructive factors such as tumours, bands, adhesions, stenosis, stricture and atresia constitute a large group of cases sometimes loosely designated "megacolon." Often cases due to organic obstruction are included in the same report with others for which no cause can be found. Indeed, many authors list obstruction as one of the main factors in the production of megacolon. This practice has been responsible for considerable confusion of thought in the literature and should, in our opinion, be abandoned. We believe very strongly that the term megacolon should be reserved for cases which are congenital and of primarily unknown origin even though certain of them, such as ours, may exhibit varying degrees of obstruction as a secondarily complicating factor.

Over two-thirds of the cases diagnosed as congenital idiopathic megacolon are infants, children and young people who have been severely constipated since birth, who go for days or weeks without bowel motion and in whom meteorism develops.

Such were the cases described by Hirschsprung[13] before the Berlin Congress for Children's Diseases in 1886.

A very few cases have been reported[27,30] in which the condition first became apparent well along in adult life, without any recognizable obstructive factor. It has been conjectured[28] that extrinsic metabolic disturbances such as avitaminosis, malnutrition, et cetera, are to blame for such deficiencies are commonly noted in these patients. It seems logical to us, however, to regard the deranged metabolism as effect rather than cause. We believe that these were probably mild cases of congenital idiopathic megacolon which for one reason or another escaped attention for years.

Other cases, likewise few in number, present an obstruction which is termed "functional" (dolichocolon) although it is really mechanical (kinking, volvulus). However, the primary disorder (elongation and hypertrophy) which makes mechanical obstruction possible is presumably related to embryonic development[25,27] and, therefore, congenital and idiopathic. Such factors are clearly seen in the patient we have presented. These are the cases that go unrecognized for years, with apparent good health and a few minor symptoms, only to develop suddenly into the classical picture of megacolon plus obstruction. Ligat[17] reported twelve which suffered from recurrent volvulus, all but one in adults. He stressed the importance of anatomical factors, elongated hypertrophied bowel and mesocolon. Eiss,[6] River[26] and Martin[20] have each reported a case of megacolon in an adult with obstruction due to volvulus or kinking, while Scott and Serenati[28] described six cases which they explained on this basis. The occurrence of obstruction is definitely a secondary complication and does not remove these cases from the congenital idiopathic category.

Detailed consideration of the numerous etiologic theories that have been advanced will be found in standard textbooks.[3,18,25] It will suffice here to mention only those few which have received most attention in the past. One explanation is that a congenital defect in colon growth results in giantism of the large bowel either before or shortly after birth. Congenital defects, however, are frequently multiple while megacolon occurs almost universally in an otherwise perfectly normal child. Achalasia of the rectal sphincter has also been blamed, but many megacolon patients have normal sphincters, mechanical dilation of the rectum often fails to produce marked improvement and spasm of the sphincter is known to occur without producing megacolon. Some authors[12] believe that lack of segmental neuromuscular coordination is responsible but no explanation for the incoordination is offered.

Due to the fact that varying amounts of improvement and relief have followed treatment directed to diverse portions of the autonomic nervous system, there has been a tendency to ascribe megacolon to a neurogenic dysfunction or imbalance. If, indeed, imbalance is the responsible factor, there is as yet no clear understanding of what produces it or exactly how it works. Interruption of the sympathetic pathway (by spinal anesthesia or sympathectomy) is found useful in some cases but not in others. The same holds true of parasympathetic stimulants (mecholyl, prostigmin, physostigmin) and parasympathetic paralyzants (atropin, syntropan).

Obviously no one common etiologic factor which will explain every case has yet been discovered. Until more light is thrown upon this perplexing problem, therefore, we are forced to accept the theory of a mixed pathogenesis.[8,25] Megacolon is a syndrome rather than a disease. As such it is possible that it may be produced by any of the factors mentioned, by a combination of them or by a factor or factors as yet unknown.

TREATMENT

In view of the high mortality (70 to 80 per cent) in untreated congenital

idiopathic megacolon, it is obvious that some attempt must be made to ameliorate symptoms and to improve, if not cure, the underlying condition. Unfortunately, there is today a wide divergence of opinion as to how this can best be accomplished.

Conservative Management. All authors are agreed that, except in those advanced cases demanding heroic emergency measures, an intensive medical regimen should first be given a thorough trial. Such management includes a low roughage diet, rectal dilatations, colonic flushes, mineral oil, vitamins, mild catharsis and meticulous intelligent supervision over a considerable period of time in an attempt to establish a normal bowel habit. In addition to these measures, drug therapy[24] is recommended in the form of parasympathetic stimulants, such as mecholyl bromide[16] and eserin[5] or paralyzants such as syntropan.[14] But there are those[15] who believe that parasympathetic stimulants have a very limited usefulness and one case of death due to drug idiosyncrasy has been reported.[16] Mild cases may respond and be carried along fairly well on this treatment. However, in most instances medical therapy, with or without drugs, will have to be continued indefinitely and immediate success must not be expected even when drugs are used.

Spinal Anesthesia. This has been employed as a therapeutic adjuvant to the medical regimen,[29,31] with beneficial results in some cases. It is also recommended as a diagnostic test[28] of use in predetermining the value of sympathectomy.

Sympathectomy. Left lumbar sympathectomy has been credited with producing striking and lasting improvement in many cases which have proven refractory to medical measures. The operative mortality is low, 2.5 per cent in one series of 117 cases collected from the literature,[22] and many do well on a carefully supervised medical regimen after operation.[12] A note of warning should be sounded, however. If the newer drugs or spinal anesthesia fail to benefit the patient, there is little prospect of improvement from sympathectomy. There is grave danger in males that sympathectomy will cause sterility (through lack of ejaculation) if performed bilaterally. In addition, bilateral sympathectomy, by interrupting pain pathways, blocks nature's warning of obstruction or impaction. Resultant perforation with peritonitis and death have been reported.[10] X-ray studies following sympathectomy consistently demonstrate that, while the condition of the large bowel may be somewhat improved, it never becomes normal, hypertrophy persists and there is always the danger of kinking, volvulus or impaction. Sympathectomy sometimes fails[10,24,33] even when done bilaterally.[32]

Ladd and Gross[15] recommend sympathectomy only in the early or moderately advanced case in which organic changes in the bowel wall are not too great. Of their six cases subjected to sympathectomy four had definite amelioration of symptoms which made further medical management much easier but two showed no significant improvement. Penick,[24] in a recent review of eleven patients (aged eighteen months to fifty-two years) upon whom left lumbar sympathectomy was performed, reported that seven had highly satisfactory results, three were greatly improved, although they still required cathartics or enemas, and only one was a complete failure. Nine of these were followed from one to eleven years. Penick recommends sympathectomy for any patient over two and a half years of age who has failed to improve under medical management but who has shown good response to spinal anesthesia or drug therapy.

In Grimson's series of twenty-four cases[10] four were subjected to sympathectomy. As a result, medical management was made easier temporarily in two and permanently in one. However, one patient died two months after operation, another had to be resected at one year because of serious impaction and a third died three years after sympathectomy of acute impaction with perforation. Rankin[25] believes that sympathectomy is the safest and surest surgical

measure for the relief of this pathologic condition. He mentions ten cases in which either bilateral lumbar sympathectomy or division of the presacral and inferior mesenteric nerves was done, with good results.

Colonic Resection. A. Subtotal resection: Certain authors[8,28,33,34,35] advocate subtotal resection of the colon in place of sympathectomy as the primary treatment of choice once the diagnosis has been established and adequate medical therapy has failed. They believe that while the operative mortality of sympathectomy is low, the operation does not appreciably alter the underlying disorder and therefore the risk of subsequent serious complications greatly outweighs the amelioration of symptoms that may be obtained.

The operative mortality of subtotal resection forty years ago ran as high as 48 per cent. Yet many then believed that the lasting benefits derived from it justified the risk involved. With the great advances that have since been made in preoperative and postoperative care, better understanding of fluid, protein and electrolyte requirements, recognition of the value of plasma and whole blood, the use of intubation for decompression and the introduction of antibiotics, chemotherapeutic agents and vitamins, the mortality rate has dropped until today it compares not unfavorably with that of sympathectomy.

In 1907 Loewenstein[19] reported 34 per cent good results in forty-four subtotally resected cases he had collected from the literature, with a mortality of 48 per cent. From 1909 to 1941 twenty-nine patients were subjected to subtotal resection at the Mayo Clinic[33] with satisfactory results in the sixteen who were followed and an operative mortality of 24 per cent. This was an overall report and does not fairly reflect improved figures for recent years. Ladd and Gross[15] reported ten subtotal resections with results as follows: five were greatly improved when last seen from one to twelve years after operation; one was greatly improved for fourteen years and then suffered a recurrence; one was improved for eight months but died of other causes. One showed slight improvement, one none at all and one died of peritonitis following operation, making an operative mortality of 10 per cent in this small series. Yeazell and Bell[34] described six children in whom a two-stage subtotal resection was done with no deaths. Five of their cases had satisfactory results.

In those few megacolon cases complicated by a secondary obstructive factor (dolichocolon) subtotal colonic resection is generally recognized as the treatment of choice although an initial cecostomy or colostomy may be required for preliminary decompression. We believe that total resection is rarely indicated as a primary procedure in these cases.

B. Radical (total) resection: Megacolon may reappear in another segment of colon years after a subtotal resection.[5,10,15,28] For this reason Grimson[10] urges radical rather than subtotal colectomy at the primary operation in advanced cases. In 1945 he reported three successive cases of radical one-stage resection with ileosigmoidostomy. Good results were obtained in all three patients up to seven, eight and twelve months, respectively, when they were last seen. Ladd and Gross,[15] on the other hand, abandoned radical colectomy after losing three patients, two from leakage at the anastomosis and one from a perforating ulcer of the sigmoid stump one year after operation. It is possible that with further improvements in surgical care and supportive treatment, radical resection may come to be more widely accepted in resistant cases. A high degree of technical skill is of course required, but the outcome in an individual case depends at least to an equal extent on the physical condition of the patient. These patients are, at best, poor risks for radical surgery and much more experience with this method is needed before it can safely be recommended for general use.

SUMMARY

The pathology, diagnosis and etiology of megacolon have been reviewed. Methods of treatment advocated by various investigators have been presented and their results analyzed. A case of megacolon in an adult has been reported.

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VARIOUS ANESTHETICS IN ORTHOPEDIC SURGERY

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THE anesthesiologist of today has a wide variety of anesthetic agents to consider in providing anesthesia for orthopedic procedures. Final selection is influenced to a marked degree by several factors. Most important of these are: first, the temperament and physical makeup of the patient; second, particular likes of the surgeon; third, the skill, ability and experience of the anesthesiologist and fourth, the particular customs of the operating room. The skill, ability, and experience of the anesthesiologist is by far the factor of greatest importance in evaluating the usefulness of various anesthetic agents. In order to utilize our present day knowledge of the various agents it is extremely important that an anesthesiologist first evaluate the patient before final decision is made as to type of agent to be employed. This evaluation is most satisfactorily accomplished by a preoperative visit to the patient, and usually with a few questions and a brief conversation an expedient method of anesthesia may be selected.

Adequate medication preceding anesthesia is desirable and important. Frequently orthopedic procedures are carried out in multiple stages so that it is all the more desirable that these patients arrive in the operating room in a tranquil frame of mind. A wise rule to follow is that all patients should receive the benefit of some type of pre-anesthetic sedation unless there is some particular contraindication. Exceptions to this rule are usually made at the extremes of life. Premedication usually consists of pentobarbital sodium gr., 1½ to 3 (0.1 Gm. to 0.2 Gm.) administered the night before operation and pentobarbital sodium, 1½ gr. (0.1 Gm.) one and one-half hours before the stated time of operation. This should be followed by doses of morphine gr., ⅛ to ¼ (0.008 Gm. to 0.016 Gm.) and scopolamine or atropine gr., 1/200 to 1/100 (0.00032 Gm. to .00064 Gm.) approximately one hour before the operation. The amount of pre-anesthetic sedation should be based entirely on information gained at the preoperative visit to the patient. Dosage of premedication should necessarily vary according to type of agent to be employed.

Morphine and Scopolamine. The production of analgesia with fractional doses of morphine and scopolamine[13] has proven very satisfactory in certain procedures such as application of a body cast or hip spica when little or no manipulation is required. If the desired analgesic effect has not been reached when the patient arrives in surgery, an additional dose of morphine and scopolamine may be administered intravenously. Employment of morphine and scopolamine analgesia facilitates many of the major plaster procedures for which complete anesthesia is not indicated.

Vinethene or Divinyl Ether. Vinethene has a limited field of usefulness in orthopedic surgery but it is valuable when a short-acting anesthetic is indicated and when profound relaxation is not required. When vinethene is employed, it must be borne in mind that the usual eye signs of anesthesia are not likely to be exhibited and that excellent analgesia usually is present when blinking of the patient's eyes is present or when there is evidence of widening of the palpebral fissures. It is in this stage of analgesia that many short manipulations of the wrist or ankle may be advantageously performed. Recovery from vinethene is prompt and is not usually attended by nausea and vomiting. This is an important factor in anesthetizing ambulatory patients since the rapid elimination of this agent permits almost immediate ambulation. Goldsmith[4] and associates and

Orth[11] and associates have demonstrated liver damage in dogs in administrations of vinethene lasting more than one hour. Martin and Rovenstine[10] have recommended that it should not be used to produce profound anesthesia or for operations requiring more than a few minutes to complete. It is our practice not to administer vinethene for procedures requiring more than ten minutes of anesthesia.

Nitrous Oxide. Nitrous oxide may be employed for reduction of certain fractures and dislocations, preferably when heavy pre-anesthetic sedation has been employed. Clement[1] uses nitrous oxide and oxygen alone for some orthopedic procedures, but for general usefulness many anesthetists combine nitrous oxide with other anesthetic agents. For orthopedic procedures nitrous oxide and oxygen combined with pentothal as described by Lundy[8] is daily proving valuable. This is a particularly advantageous combination to use when fireproof anesthesia conditions are desirable. Nitrous oxide shares with chloroform the distinction of freedom from the explosion hazard which is in marked contrast to such agents as ethylene, cyclopropane and ether. The difficulty of obtaining muscular relaxation with nitrous oxide and oxygen alone has been recently circumvented by the use of curare as an adjunct to nitrous oxide. Curare and nitrous oxide[3] and oxygen may be conveniently employed for orthopedic anesthesia problems as well as other types of surgery requiring relaxation.

Ethylene. Ethylene is used in much the same manner as nitrous oxide but should not be employed when x-ray or electrical equipment is being used.

Cyclopropane. Since the introduction of cyclopropane in 1934 by Stiles, Neff, Rovenstein and Waters,[15] this agent has enjoyed considerable popularity by both surgeons and anesthetists. Cyclopropane is the most potent of the anesthetic gases and may be employed to advantage for a number of orthopedic procedures. It has been our experience that the satisfactoriness of cyclopropane anesthesia is considerably enhanced in the majority of orthopedic situations if small amounts of ethyl ether are added to the inhaled mixture. Addition of small quantities of ether minimizes the parasympathetic effect that cyclopropane exhibits which is more pronounced in some individuals than others. Use of cyclopropane preceded by pentothal for induction has also proven very satisfactory.

Ethyl Ether. This year 1946 marks the one hundredth anniversary of the use of ether which was introduced for surgical procedures by Morton[6] at the Massachusetts General Hospital in 1846. Ethyl ether rightfully continues to carry a large share of the burden of inhalation anesthesia for orthopedic procedures. The flexibility of this agent, together with its relatively wide margin of safety, renders it a valuable agent. Ether would be even of greater serviceableness if more time and skill were devoted to its administration. Induction of ether anesthesia may be readily accomplished with vinethene, nitrous oxide, cyclopropane, ethylene or pentothal. The controllability of ether is considerably enhanced when an endotracheal tube is inserted. This is particularly so in orthopedic operations requiring placing the patient in a prone position. For example, in performing cervical laminectomy, ether has proven extremely satisfactory when administered by the endotracheal method. The value of ether for babies and small children is well established and ether anesthesia continues to be popular for children who undergo orthopedic procedures. Use of basal doses of tribromethanol with amylene hydrate (avertin) has been valuable in children on whom it is necessary to perform repeated operations under ether anesthesia. Basal dosage of 70 to 90 mg. Kg. may be employed. Avertin should be avoided, however, in operations which may prove to be of a shocking nature or operations likely to be attended with considerable hemorrhage. The incidence of using avertin as a basal anesthetic in connection with various orthopedic procedures has

been markedly reduced in recent years for two reasons: first, the increased popularity of pentothal sodium for induction of anesthesia and second, the wider application of adequate premedication.

Pentothal Sodium. The utility of pentothal is now well established in respect to the rôle that pentothal now plays in traumatic and orthopedic procedures. This in part is due to the rapid, smooth induction it usually affords. In dealing with traumatic problems a quiet induction is not only desirable but necessary many times to avoid further trauma to shattered limbs.

Anesthesiologists are constantly seeking combinations of anesthetic drugs that will provide satisfactory anesthesia with minimum doses of drugs employed. Lundy,[7] in 1928, coined the term, "Balanced Anesthesia" to refer to combinations of drugs employed to provide anesthesia whereby minimum quantities of several drugs are used. The benefit when employing this philosophy of anesthesia lies in avoiding toxic or near toxic doses of single drugs when profound anesthesia is required. Since the introduction of pentothal in 1934 by Lundy,[9] this drug has adapted itself especially well to this modern conception of anesthesia. By employing proper premedicant drugs in combination with pentothal and one of the gaseous anesthetic agents, the benefits of combined anesthesia are utilized and yet a relative wide margin of safety is assured. A popular combination for orthopedic procedures is pentothal with a 50 per cent mixture of nitrous oxide and oxygen. This combination of agents assures adequate oxygen supply yet considerable analgesia is provided by the nitrous oxide in this mixture, overcoming to some extent the inability of pentothal to provide good surface or skin anesthesia.

Pentothal sodium administered intravenously provides adequate relaxation for reduction of fractures and dislocations and for manipulations of the back and extremities. It is a standing rule in our department of anesthesia that whenever pentothal is administered, the patient is connected to a gas machine to breathe oxygen or mixtures of oxygen and nitrous oxide. This single measure has definitely increased the safety of pentothal anesthesia in our hands.

Curare. Since Griffith[5] introduced curare in 1942 for clinical use in anesthesia, the scope of usefulness of this drug has gradually increased. Curare may be administered in 20 to 40 unit doses intramuscularly to robust individuals about to receive inhalation anesthesia for orthopedic procedures. Used in this manner curare materially facilitates the induction of inhalation anesthesia. By using curare by the intravenous route it is possible to provide profound muscular relaxation with an extremely light plane of nitrous oxide and cyclopropane. For some patients this is a distinct advantage and is a method to be employed when the indications are present.

Regional Anesthetic Drugs. Procaine, pontocaine, metycaine and nupercaine are the drugs most commonly employed for regional and spinal anesthesia for orthopedic procedures. The choice of drug must of necessity be influenced by the skill and experience of the individual about to employ the drug. Procaine rightfully continues to be the drug most commonly employed for regional methods either alone or in combination with vasoconstrictor drugs. Metycaine has proven valuable when employed in a 2 per cent solution for brachial plexus block. Emergency orthopedic surgery is frequently carried out in the upper extremity of patients who have recently eaten, and the use of metycaine for brachial plexus block is many times a convenient means of providing anesthesia of the upper extremity when other methods are contraindicated. Pontocaine in our experience comes nearest to providing an all purpose agent for spinal anesthesia for orthopedic procedures of the lower extremities. It is potent and long lasting enough for most ordinary orthopedic operations. When pontocaine is combined with dextrose as described by Sise,[14] an anesthetic of definite hyperbaric character-

istics is provided and the level of anesthesia may be limited by employing the gravity technic. Prickett, Gross and Cullen[12] have recently reported the value of vasoconstrictors in increasing the usefulness of spinal anesthesia by employing epinephrine in combination with procaine. Whitacre and Potter[17] have pointed out the value of using vasoconstrictors in combination with pontocaine and nupercaine when employed for spinal anesthesia. Nupercaine may be employed for spinal anesthesia for orthopedic procedures which are destined to be time-consuming.

Refrigeration. Crossman[2] and associates have demonstrated the value of reduced temperatures in producing anesthesia of the extremities. The use of shaved ice to produce anesthesia of the extremities is well tolerated in poor risk cases and is the anesthesia of choice in desperate and moribund patients who must undergo amputation.

SUMMARY

Various anesthetic agents employed in orthopedic procedures have been briefly discussed. The value and the trend toward using combinations of anesthetic agents utilizing the beneficial qualities of several drugs has been stressed. Too much emphasis should not be placed on the anesthetic agent itself because the experience, skill and ability of the administrator is a factor of far greater importance than the particular agent employed. Recently Waters[16] has aptly stated, "It is not the tools but the way tools are used," that is the important factor in anesthesiology.

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THE CRITICALLY BURNED CHILD

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THE critically burned child presents a somewhat distinct and, indeed, a more exacting problem than does the critically burned adult. An extensive burn affects a relatively larger proportion of the child's body surface, particularly when trunk and chest are involved, since the extremities have a comparatively smaller percentage of skin area. Moreover, the child lacks the intellectual maturity essential for cooperation with the physician in the long and rigid control required for ultimate recovery. Therefore, certain specific phases in the treatment of the child patient are at variance with the therapy recommended for the adult.[1]

TREATMENT OF SHOCK

The initial step in dealing with critical burns is, of course, to win the crucial battle for immediate survival. In the author's judgment this engagement is often lost during the first twenty-four hours because the physician underestimates the oncoming shock and fails to fortify the patient with necessary systemic support. In such cases it has been found that primary or secondary shock appears from six to eight hours after inception of the burn.

Massive doses of plasma are required during this initial period, supplemented with intravenous injections or hypodermoclysis of isotonic electrolyte solutions.

PREPARATION OF WOUNDS

After shock treatment the second step is wound dressing. Whatever débridement is done before the initial dressings are applied should be carried out quickly and gently. Débridement should be kept to the minimum. The areas may be lightly cleaned with soap and water, rinsed with a physiologic saline solution and dried.

First dressings for severe burns are designed to control infection, to supply pressure that will check capillary dilatation and fluid loss and to prevent inspection and handling. The child patient is more comfortable with a dressing made of an ointment containing either an animal or a mineral base. (A 2 per cent xero form ointment is recommended.) When the time comes to remove this type of dressing, pain and bleeding are minimized. After the first twenty-four hours the administration of plasma should be discontinued as it is no longer of appreciable advantage and, in the writer's judgment, has no further place in the treatment of burns. Whole blood then becomes the life saving mechanism.

The initial dressings are left intact from ten to fourteen days. When those dressings are removed, complete débridement is done. If the burned areas fail to show healthy granulation tissue, grease dressings similar to the initial dressings are applied. These are changed every three days until granulation tissue appears. If granulation tissue is present, an alternating change of dressings from grease to wet and from wet to grease is advisable while preparing the wounds for skin coverage. The wound bacteria are resistant to local treatment and an occasional change in type of dressing provides more definite bacteriocidal action. For the same reason a change of solution is recommended for wet dressings: Dakin's, boric acid, normal saline or mild acetic acid. When wet dressings are being changed, the layer of gauze next to the wound should be removed cautiously to limit the amount of bleeding. Before the change is made all fresh dressings should be prepared for immediate application. In order to replenish the supply of blood and to bring the

hemoglobin up to at least 80 per cent, it may be necessary to give the patient whole blood. At any rate the blood should be checked during the period of wet dressing changes. The change of wet dressings each day or the change of grease dressings at three-day intervals requires a bedside routine that is a factor of some significance in handling the patient.

The surgical team is properly masked and gowned, a sterile bed sheet is spread over the foot of the bed and on it are placed tongue depressors, a bowl, scissors and two or more heavy roller bandages. When grease dressings are employed, an assistant cuts as many strips of heavy roller gauze, 4 by 8 inches, as are needed. With the use of a tongue depressor, the ointment is removed from the container and spread on the gauze. One 5 yard heavy roller bandage is placed in the bowl containing saline solution. One assistant now removes the soiled dressings and the freshly anointed strips are immediately placed over the wound. Then wet heavy roller bandage is firmly applied. Following this, come the elastic roller bandages and adhesive tape. If the granulating areas are exuberant, it is good practice to incorporate marine sponges just over the greased dressings. Incorporating the sponges in the heavy roller bandages gives them the added pressure needed to help flatten the granulating bed.

So much has been said about preparation of the wounds because a critically burned child furnishes only limited donor areas and cannot afford to lose even a small percentage of the grafts.

HOMOGRAFTING

To wait for third degree burns to close over with scar tissue is not a realistic approach. Frequently, during the third to sixth week following inception of the burn, sloughing is not complete, the granulation area appears dull and somewhat exuberant, the protein loss is high, there is a negative nitrogen balance and the hemoglobin is below 70 per cent despite several whole blood transfusions. At this period homografting may save the child's life. The third step then in the critically burned child's progress is homografting. Homografts will bridge the gap between the extreme toxic stage when autografting might prove fatal and the fourth step, which is autografting. In this mid-stage of treatment, homograft coverage seals the wounds and makes possible the attainment of a positive nitrogen balance, an increase in food intake and an improved blood picture. Naturally, autografting must be planned from the outset and the wounds must be treated with this purpose in mind.

Repeated homografting to await scar coverage is a false hope if the burned areas are extensive. When the grafts liquefy, only a small part of the burned areas may be flooded over with scar epithelium. Circular areas seldom close over. In the meantime the patient's morale is seriously affected. Gain in weight or even maintenance of body weight is extremely difficult; the nitrogen imbalance grows; debilitation becomes more pronounced and fatal termination threatens.

Experience indicates that a second coverage of homografting from the original donors fails to "take" and skin from new donors adheres for a lesser time than did the original donor take. For although the first sheets of skin may remain viable for from three weeks to two months, the second crop remains viable for no more than two weeks. It is a ticklish question as to how many times homografting should be repeated. In the author's experience, in the case later to be described, homografts were made twice.

Up to this point no mention has been made of the food intake of the critically burned child. From the beginning he should be placed on a high protein, high vitamin diet but frequently he will refuse food, nor will his resistance collapse under persuasion be it gentle or firm. He probably finds amino acids distasteful, in which event tube feeding is the sole recourse. Amino acids are to be prescribed as a daily supple-

ment to the regular food intake, 4 Gm. per Kg. Eggnogs, milk and cream and broths with a minimum amount of salt, to prevent edema, also should be given. Administration of multiple vitamin concentrates is suggested.

In preparation for the first homografts, supplemental feedings of amino acids are carried on for a week or ten days in advance of surgery. A thick mixture (1 ounce to each 20 pounds of body weight) is placed in a large ear syringe and forced into the feeding tube. The child should be placed in a semisitting position in order to prevent vomiting, as the amino acids are sent through the tube. No water is permitted for one-half hour before or after the feeding. Tube feeding is continued for two weeks after either homografting or autografting.

One week before grafting the grease dressings are discontinued and only wet dressings are employed. The dressings are wet every three hours day and night with daily changes of dressings. Saturation of the dressings is necessary to prevent extreme pain and free bleeding when they are removed next day. On the day of surgery the dressings are changed early in the morning. Postoperatively the wet dressings are continued for six days. Thereafter, between grafting stages ointment dressings are reemployed to add to the child's comfort. During the homografting process, 250 cc. of whole blood is administered postoperatively at the bedside; this is repeated on the third day.

The initial homografts are done as quickly as the granulating tissue is reasonably clean, usually three or four weeks after the burn. To obtain the greatest percentage of coverage it is advisable to suture the homografts under moderate tension. If a general anesthetic is contraindicated, then the homografts can be brought back to the original size and tension by glueing one side of the graft on gauze and stretching and glueing it down to the opposite side. Immediately afterward the graft with the attached gauze is placed on the wound. The same procedure is followed with each homograft taken from the donors.

The covered wounds are then snubbed on with two or more heavy roller bandages followed by an elastic bandage and adhesive plaster. Two or three weeks later if the patient has gained weight, if the red cell count is 4,000,000 and if the hemoglobin exceeds 80 per cent, the patient can be prepared by daily changes of wet dressings for the first stage of autografting. As has been indicated an additional homograft or two may be necessary before the child is physically fit for the autografts.

AUTOGRAFTING

When the child is ready to begin the final stage, permanent coverage of his wounds, the routine of tube feedings, whole blood administration and the application of wet dressings, as previously described in the homografting process, must be repeated. Sometimes there is a problem in skin grafting as to the sequence of events: Should the graft be taken from the patient before or after the wound is prepared? When dealing with a contaminated area, such as a burn wound, it is a sound principle to start from the clean site and advance to the contaminated site; therefore, in all burn wounds the grafts should be taken first.

Because infection is ever present, penicillin is used routinely. During surgery the proportion of 100,000 units per 5 cc. of saline solution is injected beneath the grafts at several points with a fine needle. Streptomycin, 100 mg. per cc. of saline solution, is added when the culture shows Bacillus pyocyaneus.

The child with an extensive burn provides only limited areas from which grafts can be had for the coverage of wounds. With donor sites limited, it is necessary that the first graft be taken no thicker than 0.006 or 0.008 inch in order that a second and a third crop can be garnered later. Nor is it advisable to subject the child to repeated trips to surgery by removing only small amounts of skin at each

operation; instead, as many as six or eight drums of skin should be taken at one time.[2]

When after intervals of a month or so the second and the third grafts are taken from the same sites, the dermatome should be set at 0.008 inch as the epithelial layer is exceedingly thin and friable. The surgeon must resist the temptation to increase the thickness beyond 0.008 inch on the second and third drums as there is danger of picking up all of the subepithelial elements. To get the requisite thinness of 0.006 or 0.008 inch a dermatome is used. The brush employed to apply the cement to the drum and to the donor site is placed in ether until it is needed, at which time the ether is shaken off. The glue must be thin when applied to the donor site and drum and the surgeon must pause until the glue is entirely free from sheen. The cutting blade, which is held at calibrated distances from the drum, is tested with "shims" of 0.006 and 0.008 inch. The shim is a unit of measurement similar to the one used by automobile mechanics in testing distances of tappets.

The late Dr. Earl C. Padgett, when the dermatome was first manufactured, suggested to the author the idea of double checking with shims. This test will frequently reveal a difference of from 0.002 to 0.004 inch at either side of the starting point of the drum although to the naked eye both sides appear equal. Another such test can well be made at the halfway spot on the drum so that, if necessary, the calibration can be reduced one or two notches before proceeding to the end of the drum. This provision is particularly needed if grafts are to be taken from the inner aspects of the thigh and from the abdomen as the skin in these areas is much thinner than is supposed.

Removal of the graft from the drum is made with extreme caution as it will tear readily. Upon its removal the graft is placed over a granulating area and the epidermis is immediately covered with a sulfonamide powder to nullify the adhesiveness of the glue.

Small grafts are not favored; if placed unsutured, many of them slip away whereas to suture them, dangerously prolongs the child's surgical experience. If stamp grafts or other grafts are applied without normal tension, from 20 to 30 per cent of their potential is actually lost. If instead a graft 4 by 8 inches is cut, it can be stretched to 4 by 10 or 12 inches by means of a new type of massive autografting which will be described.

The graft is basted on one side while one of the surgical assistants stretches it under moderate tension; meantime the surgeon cuts large slits in the graft longitudinally or vertically to provide added width or length. By this method the area has been given the fullest amount of skin coverage and the take of the graft extends over a greater area. Flooding over by epithelium into the interstices occurs rapidly, within two weeks. As soon as the grafts are taken the donor area is sprayed with thrombin solution to limit blood loss at the donor site.

It has been estimated that following the removal of a split graft of approximately 30 square inches the donor site will lose as much as 50 cc. of blood. When four or five drums or more of split skin are removed at one operation, the amount of blood loss is a serious consideration. The child's susceptibility to shock, plus the blood loss from donor sites, necessitates the use of large amounts of whole blood beginning in the surgery and carried on for four days postoperatively. As much as 750 cc. may be necessary for the first twelve hours for a child four or five years of age. Donor sites will stop losing proteins in four days and will heal over with epithelium in six or seven days.

The following case report demonstrates application of the measures described on a child with very extensive burns.

CASE REPORT

M. S., a boy of four, suffered extensive third degree burns on May 27, 1946, when his flannel pajamas caught fire while he was playing with matches. (Figs. 1 and 2.)

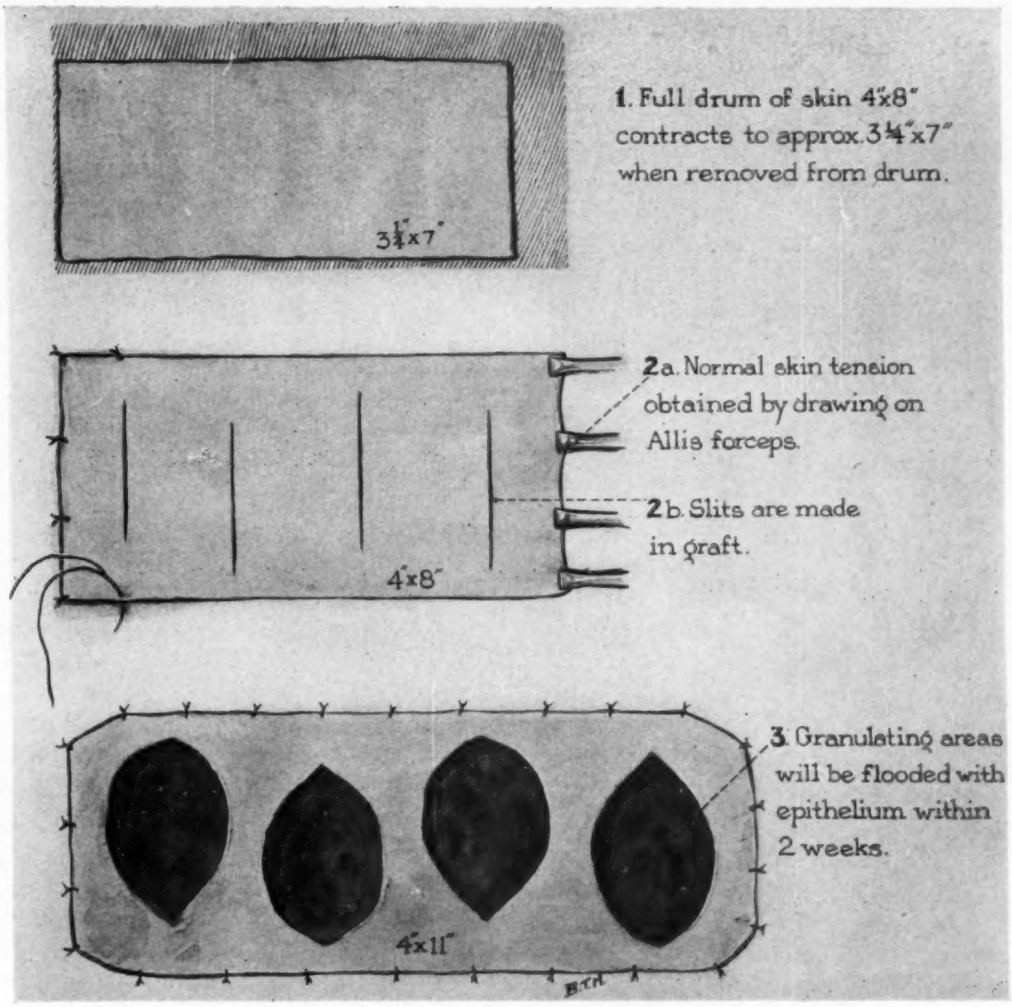
1. Full drum of skin 4"x8" contracts to approx. 3¼"x7" when removed from drum.
3¼"x7"
2a. Normal skin tension obtained by drawing on Allis forceps.
2b. Slits are made in graft.
4"x8"
3. Granulating areas will be flooded with epithelium within 2 weeks.
4"x11"

Fig. 1.

When first seen at Illinois Masonic Hospital, Chicago, six weeks after the burn, the affected areas showed numerous patches of necrotic skin and exuberant granulating areas. Removal of the dressings resulted in free bleeding. Following a two weeks' regimen of daily change of dressings (firm wet heavy roller bandage), of tube feeding with high protein concentrates and of several whole blood transfusions, the patient was taken to surgery.

Four full drums of thin split-thickness skin were taken from the mother and father and sutured under normal tension. Two dermotome drums of thin split-thickness skin were taken from the inner aspect of the patient's thighs to cover the right elbow and axillary region.

The second surgery was delayed eight weeks owing to an acute hepatitis and acute nephritis. The icteric index climbed to 80 but returned to normal under daily intravenous administration of 50 per cent glucose solution. At this surgery seven full dermatome drums of thin split-thickness homografts were snubbed on the wounds without an anesthetic.

The third surgery was six weeks later when a second crop was taken from the inner aspects of the patient's thighs and one dermatome drum from the outer aspect of the patient's thigh. After another six weeks' interval came the fourth surgery. Five dermatome drums of thin split-thickness skin were taken from the patient's abdomen, the gluteal regions and the outer thighs. Four weeks later the patient again submitted to surgery, at which time four dermatome drums of thin split-thickness skin were taken from his abdomen, the gluteal regions and the outer thigh.

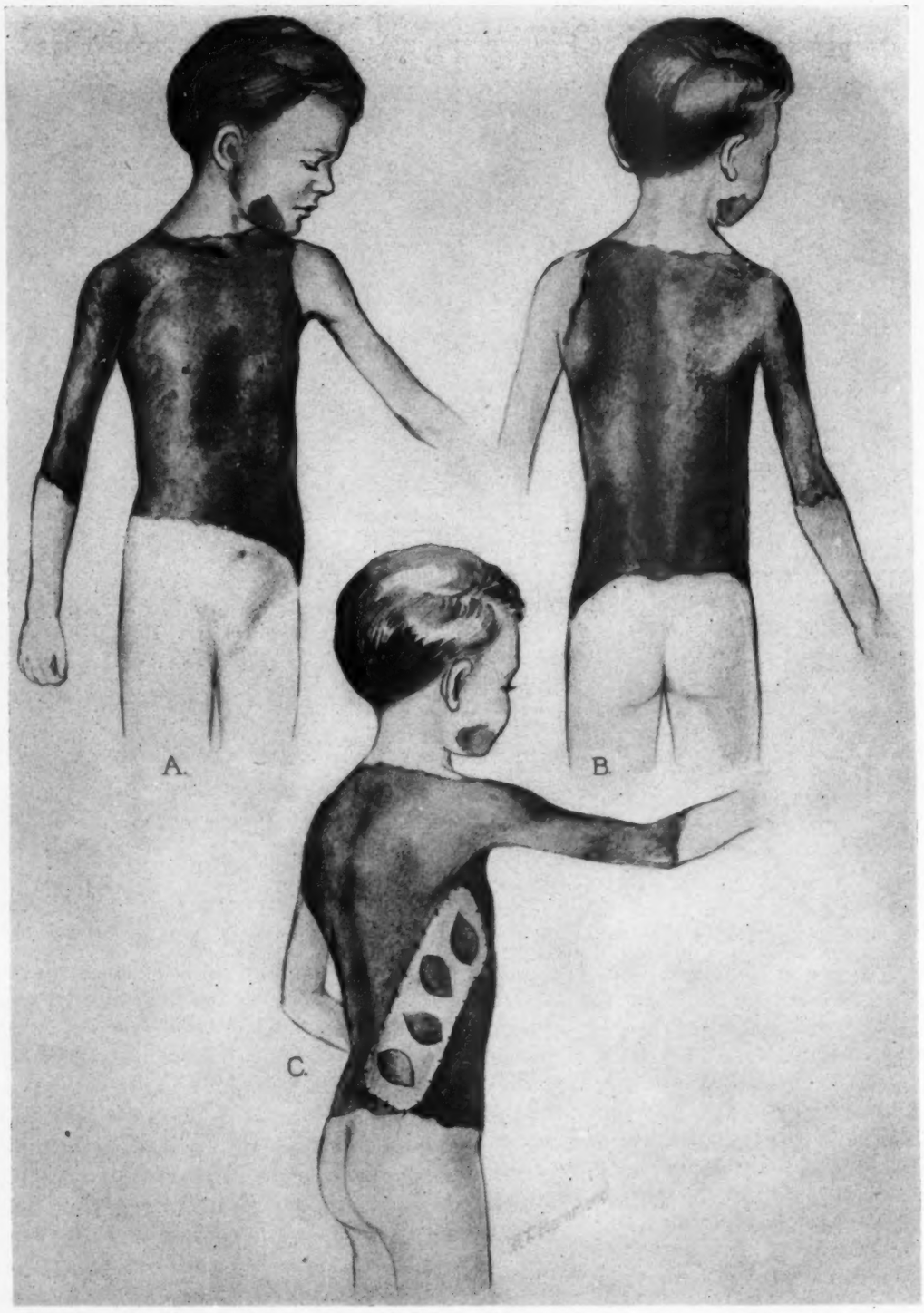

FIG. 2. A, third degree burn front; B, third degree burn back; C, full drum of skin 4 inches by 8 inches sutured to wound; slits made in graft.

Thus eleven full drums of homografts and fourteen drums of autografts, or a total of twenty-five drums of skin, resulted in complete coverage.

SUMMARY

In summary, the following points might be reviewed:

1. In the case of the critically burned child prepare to modify some of the technics recommended for adults.[1]

2. Anticipate primary or secondary shock from six to eight hours after inception of the burn. Give massive doses of plasma and electrolytes to correct circulatory imbalance.

3. Discontinue plasma after twenty-four hours and substitute whole blood.

4. As soon as granulation of wounds begins alternate the dressings between grease and wet.

5. If the patient is listless, is deficient in nitrogen and has a low hemoglobin, institute homografting preliminary to autografting.

6. Perform autografts with the thinnest possible thickness of skin (0.006 to 0.008 inch) in order to obtain rapid healing of donor sites so that removal of a second, third or fourth crop from the original donor sites can be done without too great an interval between surgical interventions.

7. Take massive autografts to cut down subsequent autografting. A new system of slitting large grafts gives increased coverage.

8. Organize teams for both taking grafts and applying grafts. A three-man team is needed for the application of the grafts. The patient should not be under surgery for more than one hour.

CONCLUSION

Homografting may become a life saving measure in the treatment of the critically burned child and represents a logical step in the preparation of the child for ultimate coverage of wounds with autografts.

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RICHTER'S HERNIA*

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RICHTER'S hernia is a form of strangulated hernia in which less than the full circumference of the bowel is caught by the hernial ring. (Fig. 1.) The terms "small hernia"[1] and "partial enterocele"[3] are found in the literature. Richter's original discussion,[1] probably first published in 1778, was the first to segregate this type of hernia although it is not clear that strangulation was considered necessarily a part of the picture. This variety of hernia is surgically urgent because gangrene occurs much earlier than in ordinary strangulations. Watson[13] states that "this is undoubtedly due to direct pressure exerted on the intestine by the constricting ring. In ordinary enterocele the mesentery or omentum, on account of its elasticity, acts as a cushion or buffer and this delays the onset of strangulation." Richter's hernia may be difficult to diagnose because of its small size, because the pain is often poorly localized and minor in character and because obstructive symptoms are extremely variable. The latter may vary from obstipation to diarrhea. Occasionally bowel movements are perfectly normal. Differential diagnosis of Richter's hernia through the femoral "empty space" is often difficult since it may not be distinguished from enlargement of the lymph nodes in this region. They are both deflected upward by the extension of Scarpa's fascia down the thigh which blends with the deep fascia

FIG. 1. *Anatomical Study*—Femoral Hernia: Poupart's ligament has been divided. Scarpa's fascia shown blending with deep fascia of thigh 3–4 cm. below Poupart's ligament. This barrier completely covers the fossa ovalis thus directing the hernia upward.

* From the Second (Cornell) Surgical Division, Bellevue Hospital, N. Y.

some 3 to 4 cm. below Poupart's ligament. (Fig. 1.)

The patients studied here were admitted to the Second (Cornell) Surgical Division between December, 1917 and July, 1945. We have included only those in whom the surgical disorder was definite and unmistakable.

ANALYSES OF PATIENTS

Age. The average age of these patients was sixty-one years. The hernias occurred most often in those in the sixth and seventh decades; (Fig. 2) the youngest patient was forty-one, the oldest eighty-nine. Fowler[3] states that the condition "appears to be limited to adults." Gouvernale et al.[9] state that the condition is seldom seen in children but mention Rhodes' case of a baby three weeks old. Patients eleven years of age and under were not admitted to our service but we noted a significant lack of young adults.

Sex. The exact ratio of females to males is not clearly stated in the literature. It is said[3] that hernias of this type are more common in females. Another author[9] goes so far as to say that females outnumber males by three, or two to one. In our series there were nine males and eight females. These figures are weighted by the fact that the Bellevue Hospital surgical population is predominantly male, there being two male wards to each female.

Site of Hernia. It is stated[1] that this type of hernia can occur at any body orifice. Treves,[2] citing fifty-three cases, noted thirty-eight femoral, thirteen inguinal and two of the ventral variety. The femoral variety makes up 90 per cent of all Richter's hernias according to Gouvernale.[9] In our series fifteen were of the femoral variety and two were inguinal. Both inguinal cases occurred in males.

Contents of Sac. The clinical size of the sac varies considerably depending on the amount of peritoneal fluid present and whether other structures, e.g., the omentum or appendix are present. It can be very small and in one of our patients Case XIII the presence of a tiny inguinal mass was first discovered in the operating room during abdominal preparation. When muscles relax under anesthesia, the involved bowel frequently slips back into the abdominal cavity and in such a case it may be impossible to retrieve or see the loop through the hernial opening. In such cases laparotomy is always performed. The ileum is the most common portion of the bowel involved[9] but the colon and jejunum are also found to be common sites of involvement. In all of our patients small bowel was found. In ten cases the bowel was viable and after a suitable period of observation was returned to the abdomen. Six patients showed gangrenous gut and one presented gangrene with perforation and the formation of a fecal fistula. Duration of obstructive symptoms in these seven patients with gangrene averaged just over nine days, the shortest was four and the longest was twenty-one days. When gangrene was absent, obstructive symptoms averaged 4.25 days; the shortest was of twelve hours' duration and the longest fourteen days. In two patients incarcerated omentum was found. The sac contained bloody fluid in eight and clear fluid in five patients. The presence of fluid was unrecorded in four cases.

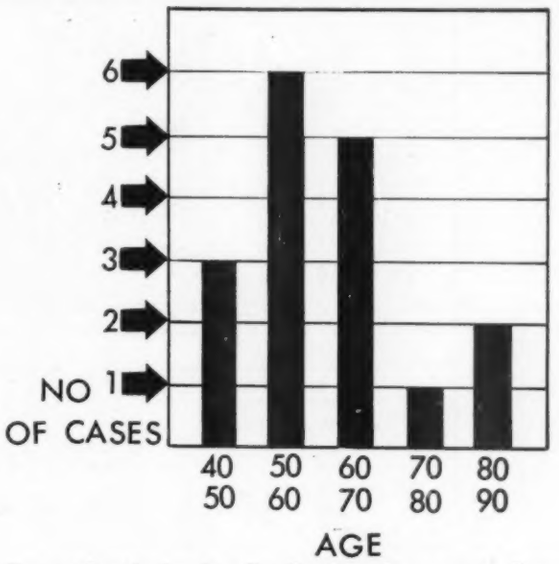

Fig. 2. Graph showing distribution of cases according to age.

Table I
Summary of Cases

Case No.	Hosp. No.	Date of Admission	Initials	Sex	Age	Previous Hernia	History	Side	Type	Contents of Sac	Operative Procedure	Choice of Anesthesia	Results
I	11-D2	12-1-17	A. F.	M	82	None	Abdominal pain, vomiting, obstipation for 2 weeks	L	Femoral	Omentum, and small bowel bloody fluid	Femoral incision, catgut repair	Local	Wound infection, bronchopneumonia, died 40th postoperative day, no autopsy
II	21-D2	5-8-21	H. Y.	M	53	Many yr.	Incarceration for 4 days; vomiting, obstipation for 2 days	L	Femoral	Small bowel, bloody fluid	Femoral incision, catgut repair	General	Uneventful course; follow-up four months; no recurrence
III	23-D2	6-23-21	W. C.	M	41	5–6 yr.	Sudden effort resulting in incarceration; vomiting, obstipation for 12 hr.	R	Femoral	Small bowel, bloody fluid	Vertical incision crossing Poupart's, inspection of bowel in abdomen; Bassini and femoral repair, catgut	General	Uneventful course; discharged healed; no follow-up
IV	58-D2	2-17-23	B. K.	F	47	10 yr.	Abdominal pain, anorexia, nausea and vomiting for 1 day	R	Femoral	Small bowel, clear fluid	Femoral incision, catgut repair	Local	Uneventful course; no recurrence in two months
V	69-D2	5-21-24	J. S.	M	68	None	Mass appeared in inguinal region 3 weeks before admission; drained feces 1 week	R	Femoral	Fecal fistula from 1 inch defect in small bowel strangulated in femoral ring	Right lower split rectus incision small bowel reduced; defect repaired in 3 layers, abdomen drained	General	Sinuses on thigh healed two weeks after two segments of fishbone were discharged; no recurrence
VI	78-D2	11-5-26	J. K.	M	58	None	Inguinal pain on exertion 2 weeks, abdominal pain, vomiting, obstipation 3 days	R	Femoral	Gangrenous small bowel ¾ circumference	(1) Femoral incision extended on to abdomen, gangrenous bowel exteriorized; (2) gangrenous bowel excised end-to-end anastomosis with Murphy button	1. Local 2. General	Passed button twelfth day; wounds healed; recurrence upper end incision one year
VII	39-D2	2-2-28	J. P.	M	67	8 yr.	Abdominal pain with mass in right groin 1 day	R	Femoral	Small bowel with bloody fluid	Femoral and right lower rectus incisions; catgut repair of femoral defect	Spinal	Paralytic ileus and bilateral bronchopneumonia; died 3rd postoperative day
VIII	53-D2	1-26-34	K. B.	F	69	None	Abdominal pain, distention, vomiting and obstipation, 6 days	R	Femoral	Gangrenous small bowel ⅓ circumference	Femoral incision, gangrenous area excised; transverse repair; catgut repair of hernia	Spinal	Paralytic ileus and bronchopneumonia; died 1st postoperative day
IX	14163-35	3-25-35	L. O.	F	52	None	Abdominal pain, vomiting and obstipation, 6 days	L	Femoral	Small bowel, clear fluid	Femoral incision, catgut repair	General	Uneventful course; no recurrence in two years
X	43481-35	9-8-35	M. F.	F	59	1 yr.	Abdominal pain, vomiting and obstipation, 8 days	R	Femoral	Small bowel, clear fluid	Femoral incision, catgut repair	Spinal	Paralytic ileus; bronchopneumonia; died postoperatively 1st day
XI	2532-37	1-14-37	S. P.	M	70	20–30 yr.	Abdominal pain, vomiting and obstipation, 4 days	R	Femoral	Gangrenous small bowel ½ circumference with bloody fluid	Femoral incision, bowel resected Murphy button anastomosis, abdomen drained	Local	Died 1st postoperative day; pulmonary edema

TABLE I (*Continued*)

Case No.	Hosp. No.	Date of Admission	Initials	Sex	Age	Previous Hernia	History	Side	Type	Contents of Sac	Operative Procedure	Choice of Anesthesia	Results
XII	4687-37	2-28-37	A. C.	F	58	None	Abdominal pain, vomiting and obstipation, 2 days	L	Femoral	Small bowel with clear fluid	Femoral incision, catgut repair	Local	Uneventful course; no recurrence 1½ year later
XIII	19221-43	4-21-43	R. B.	F	45	None	Abdominal pain, vomiting and obstipation, 5 days	R	Femoral	Gangrenous small bowel, bloody fluid	Right rectus incision; resection small bowel; end-to-end anastomosis	General	Paralytic ileus; bronchopneumonia; pulmonary edema; died first postoperative day
XIV	47515-44	10-18-44	A. H.	M	64	1 yr.	Abdominal pain, vomiting and obstipation, 2 days	L	Femoral	Small bowel, clear fluid	(1) Left rectus incision, hernia reduced; (2) femoral incision, catgut	1. General 2. Local	Uneventful course; no recurrence one year
XV	14387-45	3-21-45	W. E.	M	58	None	Abdominal pain, vomiting and obstipation, 4 days	R	Inguinal	Small bowel	Inguinal incision, reduction, Halstead repair with catgut	General	Uneventful course; recurrence in ten months
XVI	26922-45	6-5-45	A. S.	M	65	5–6 yr.	Incarceration of right inguinal hernia 1 week	R	Inguinal	Gangrenous small bowel and omentum, bloody fluid	Femoral incision, gangrenous bowel retracted into abdomen; incision drained, low midline incision. Gangrenous bowel resected end-to-end anastomosis	Spinal, General	Uneventful course; no recurrence in seven months
XVII	31990-45	7-5-45	L. S.	F	89	None	Abdominal pain, vomiting and obstipation, 1 week	L	Femoral	Gangrenous small bowel and omentum, bloody fluid	Femoral incision, resection small bowel, end-to-end anastomosis Murphy button	General	Uneventful course; passed button 15 days; discharged well

Fig. 3. Artist's sketch showing technic of bimanual reduction of hernia.

History of Previous Hernia. In nine cases the patient was unaware of any difficulty until the onset of acute symptoms. The remaining eight patients had vague histories of known hernias ranging from one to thirty years in length of time.

Treatment. The treatment is prompt operation, the earlier the better. However, if the patient's condition is poor due to obstructive symptoms, time is taken to restore fluid and electrolyte balance. Closed reduction is never attempted. No operative routine is suggested as each patient presents a different combination of problems. Only two things are of prime importance: (1) to reduce the hernia and (2) to be assured of viability of the strangulated bowel. Elegant hernia repair can wait until a later operation unless the patient's condition allows it.

A glance at the column entitled operative procedure in Table 1 reveals a number of ways of handling this problem. With minor variations they fall into three groups: (1) Those handled by an incision directly into the hernial sac, inspection of bowel, resection if necessary and hernial repair (ten cases). (2) Those handled by laparotomy at a distance from the hernia, reduction and inspection of bowel with resection if necessary (four cases). (3) Those handled by two incisions or by a vertical incision across Poupart's ligament (three cases).

The authors believe that the last method is to be avoided if possible. The making and repair of two incisions is time-consuming and frequently unnecessary. The need for a second incision arises for three reasons: (1) The bowel reduces and cannot be retrieved for inspection through the hernial opening or (2) the mesentery is too short to allow withdrawal of sufficient bowel for inspection or repair or (3) the

hernial opening is too small to allow adequate bowel to be withdrawn. In our experience a lower rectus incision followed by reduction is advisable in certain cases. A combination of pressure over the hernial mass and traction on the strangulated loop from within the abdomen is safely done. (Fig. 3.) The mesentery relaxes, blood supply is aided by hot packs and after fifteen minutes or so very doubtful bowel wall will begin to show peristalsis. If resection is necessary, it can be done under optimum conditions. The hernia may be repaired intra-abdominally as suggested by Cattell[5] or be left for routine elective repair later on.

If the hernia is clinically small and if gangrene is probable, then we believe this approach to be mandatory. The penalties following improper anastomosis done under difficulty or those following return of nonviable gut to the abdominal cavity remain enormous. The penalties of an accidental rupture of gangrenous bowel in the manipulative reduction have become progressively smaller due to advances in chemotherapy. If the hernia is large or recent and if one is reasonably sure gangrene is not present, routine reduction and herniorrhaphy should be attempted.

RESULTS

The mortality in our series was six patients in seventeen or a little better than one in three. It is interesting to note that three of these deaths occurred in patients in whom viable bowel was found. The mortality of patients requiring resection was 50 per cent. Four patients died with paralytic ileus and one died on the fortieth postoperative day following wound infection and bronchopneumonia. The remaining death ensued due to cardiac failure with pulmonary edema.

Our late follow-up is incomplete. Three patients never returned following discharge. The other eight were followed for a period of from two to twenty-four months. Recurrent hernias were discovered in two patients.

SUMMARY

Seventeen cases of Richter's hernia are presented. Anatomic considerations and methods of treatment are discussed.

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Case Reports

INTERMITTENT RAYNAUD'S PHENOMENON RESULTING FROM NON-UNITED FRACTURE OF THE NAVICULAR BONE

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ACCORDING to Allen, Barker and Hines,[1] "Raynaud's phenomenon may be defined as episodes of constriction of the small arteries and arterioles of the extremities resulting in intermittent changes in color of the skin of the extremities such as pallor, cyanosis or both. Raynaud's phenomenon may occur primarily as in Raynaud's disease or it can occur secondarily in association with a number of conditions and diseases."

The present communication deals with Raynaud's phenomenon on a traumatic basis and resulting from a non-united fracture of the navicular bone. It is presented to call attention to a somewhat unusual complication resulting from fracture of a carpal bone. It may further afford an opportunity for considering one type of mechanism precipitating Raynaud's phenomenon under conditions simulating an experimental setup as presented by a simple, well localized form of human pathologic condition.

CASE REPORT

A Norwegian seaman, forty-four years of age, was first seen by the author in August, 1941. His main complaint was pain in the left hand which he stated dated back to 1919. When then in the navy, he fell one day and hit the dorsum of his left hand against the floor. Subsequent x-ray revealed a fracture of the scaphoid bone. A plaster cast had been applied and worn for two or three weeks. The patient was declared unfit for navy service during the next six months. Upon returning to his former duties, he noted pain in the wrist of his left hand. During the first few years this pain was bearable and did not prevent him from performing his work. However, it bothered him, sufficiently to make him seek medical advice on several occasions. Physiotherapy and massage had a temporary effect.

During the last four years the pain in his left wrist had greatly increased in severity. Consequently, he had been able to carry out his work aboard ship with only the greatest difficulty. During the last two years additional symptoms had been observed; when performing certain functions involving the left hand (e.g., lifting a heavy trunk with this hand), he would note the onset of a tingling sensation of the fingers of this hand, quickly followed by severe pain in the wrist and entire hand, and later a sudden feeling of weakness in the muscles of the hand which would result in the dropping of anything he might be carrying in this hand. During these episodes the fingers of the left hand would appear "completely white and dead," and would feel cold to the touch. A pronounced numbness of the volar sides of the fingers would be experienced. After a few minutes the white color of the fingers would change to bright red or bluish red. Simultaneously he would note a sudden sensation of local heat. The superficial sensitivity and the mobility and strength of the hand would then gradually return. It could not be ascertained if these attacks had any relation to the environmental temperature as they occurred just as frequently during summer as during winter. They were not precipitated by worry or anxiety alone. Similar changes in the right hand or in any other part of the body had never been experienced. The patient also had noted a less

severe pain radiating from the left wrist to the lateral side of the elbow. This pain always occurred with the previously described attacks; however, it would also be present independent of the attacks. Almost every day during the last year he had felt a sore ache around the lateral left epicondyle which was very tender to pressure. Occasionally, he also had noted a moderately large, tender, swelling around the left epicondyle. The patient had grown increasingly nervous and mentally exhausted during the last year due to his physical sufferings, and to the anxiety of life aboard a ship that was constantly travelling through U-boat infested waters.

Physical examination revealed that the patient was well developed and of powerful musculature. He appeared tired and nervous with moderate tremor of the fingers and of the tongue. The thyroid gland was not enlarged. The basal metabolic rate was within normal limits. His blood pressure was 130 mm. Hg systolic and 90 diastolic, with a pulse rate of 74 beats per minute.

Local examination revealed normal mobility of the left wrist and fingers except for moderate inhibition in radial flexion of the wrist. No definite motor or sensory changes were found. Palpation of the left wrist revealed a bony, hard irregular prominence in the bottom of the "snuff box." It was very tender to direct pressure and moderately mobile. Pain in this arm could also be elicited by radial deviation of the wrist joint. This pain would radiate along the radial side of the forearm to the lateral epicondyle of the elbow. Palpation of the left radial and ulnar arteries revealed normal pulsations as was also found in the other peripheral arteries. Physical examination otherwise was essentially negative. Routine laboratory investigations, including the Wassermann reaction, revealed nothing of particular significance.

X-rays revealed a transverse, non-united fracture of the left scaphoid bone. (Fig. 1.) The distal fragment showed a bulging of its anterior, external portion which impinged against the external portion of the articular surface of the radius. This had caused a bony lip formation of the styloid process of the radius at the point of contact with the fragment.

In an attempt to reproduce the previously described attacks of pain and other phenomena the following investigations were undertaken: Insertion of the patient's left hand in ice water (8°c.) for ten minutes failed to produce any changes different from that of the right hand, and without any indication of Raynaud's phenomenon. Alternate closing and opening of the left hand for five minutes at a time, with digital pressure compressing the ulnar and radial arteries separately as well as simultaneously, failed to produce any other symptoms than a feeling of muscular tiredness in the hand. By carrying and manipulating a weight in his left hand (a 16 pound typewriter) the patient soon experienced a severe pain in his left wrist; this was associated with a tingling in and pronounced pallor of the fingers. The tactile sensitivity on the ulnar aspect of the second to the fifth finger and over the dorsum of the distal two-thirds of the same fingers was definitely impaired. Simultaneously, the strength of his left hand, as tested by grip of the hand, was greatly impaired as compared to its strength before the carrying of the weight. The pallor persisted for almost one minute after release of the weight and was followed by a sensation of increased local warmth and rubor. Palpation of the pulsation of the radial artery during the stage of pallor indicated a definite decrease although not complete cessation of pulsation. The above experiment with the carrying of the weight in the left hand was then repeated after injection of 8 cc. of 1 per cent

Fig. 1. X-rays of the left wrist before operation.

Fig. 2. X-rays of left wrist after operation.

solution of novocain around the palpable movable fragment and around the radial artery of the snuff box. During this experiment the patient did not complain of pain. Likewise, the Raynaud's phenomenon could not be elicited.

Operation was performed on August 24, 19[illegible] using local infiltration with 1 per cent novocain. A 3-cm.-long incision was made in the left snuff box and parallel to the ulnar aspects of the long extensor pollicis tendon. The radial artery was readily located; this was carefully exposed; its wall appeared normal with normal pulsation throughout the exposed segment. A careful palpation of this segment did not give any suggestion of thickening of the wall of the artery nor of the presence of intra-arterial thrombus formation. With full extension of the wrist and fingers, the movable fragment of the navicular bone could hardly be felt. Upon maximal ulnar flexion of the wrist, this movable fragment was felt to protrude distinctly and was seen to impinge gently against the posterior wall of the artery. This impingement did not result in any visible or palpable changes in the pulsation or the caliber of the artery. Raynaud's phenomenon of the fingers was not seen to ensue. By gently pulling the artery and the tendons to one side the movable fragment of the navicular bone was readily freed and removed.

An area of numbness at the base of the left thumb was noted immediately after the operation; otherwise, there were no sensory or motor nerve disturbances. The postoperative course was uneventful with primary healing of the wound. Two days after the operation it was noted that the previously observed local tenderness about the lateral epicondyle of the left elbow had disappeared. On the same day the patient stated that the subjective symptoms of pain in the elbow had disappeared. (Fig. 2.)

Because of the patient's continuous service aboard ship during the war, he was granted a convalescence of five weeks before returning to his ship. During this period further observations with or without carrying a weight in his left hand revealed return of full and normal mobility without pain. Significantly, the Raynaud's phenomenon as observed before operation could not be produced.

Since the patient signed aboard ship again, he has been seen by the author on several occasions, the last time in February, 1946. According to him, he never thinks about favoring his left hand any more and considers it as strong and as useful as before the accident in 1919. Since dismissal from the hospital, no further Raynaud's phenomenon has appeared. Repeated investigations of his peripheral vascular tree have not revealed the presence of any peripheral vascular disease.

COMMENTS

The primary lesion in the present case appears clear enough, namely, a non-united fracture of the left navicular bone of more than twenty years' duration.

The question may be raised: did the patient, aside from the fracture, have a primary peripheral vascular disease? Without taking up a detailed discussion of this point, I believe the report as presented includes essential information sufficient for the conclusion that any type of primary peripheral vascular disease did not exist. The local peripheral vascular phenomenon is therefore concluded to have taken place on the basis of the existing non-united fracture.

How was the Raynaud's phenomenon produced in this case? This seems a simple enough question. To students of the physiopathologic conditions of peripheral vascular diseases Raynaud's phenomenon, however, represents one of great controversy. This is well illustrated by the present conception of the physiopathologic disorder of Raynaud's disease. According to one school of thought, the essential abnormality is to be found in the vasomotor system, this system being abnormally stimulated by extrinsic factors (cold) resulting in hyperactivity of the vasoconstrictor fibers. Opposing this view is the conception that the vasoconstriction primarily is the result of changes in the vessel wall itself.

Without taking up this controversy of the mechanism of Raynaud's phenomenon on a general basis, it may be permissible briefly to discuss it in relation to the present observations. Cold is generally accepted as one of the most common factors eliciting or aggravating Raynaud's phenomenon; this fact naturally does not indicate whether or not the abnormal changes are present primarily in the vasomotor fibers or in the arterioles themselves. It only indicates that the nerve fibers or the arterioles are in a pre-activated state and respond with constriction upon stimulation by low temperature. In the case herein reported the cold factor failed to stimulate the vessels to spastic contractions; in other words, the vessels acted as if no preliminary state of hyperirritability of nerve fibers or arterioles existed.

"Working" of the hand with digital compression of the radial artery failed to produce Raynaud's phenomenon. This would indicate that the phenomenon was not produced by intermittent occlusion of the radial artery itself. Further support for this view was gained by the observations at the time of operation that even with maximal ulnar deviation of the wrist the loose fragments of the navicular bone in no way did result in segmental compression of the radial artery. The logical inference from the latter observation is that we are confronted with a true intermittent constriction of the arterioles distal to the level of the fracture fragment.

Severe pain as well as the typical Raynaud's phenomenon was reproduced in the patient when he carried a relatively heavy weight in such a manner that alternate ulnar and radial flexion would take place in the wrist. From observations made at the time of operation it is possible in retrospect to reconstruct what was taking place during the act of weight carrying. The loose navicular fragment would alternately be expressed and depressed. During its stage of protrusion it would be forced up against the posterior wall of the radial artery. Soon severe local pain and Raynaud's phenomenon would ensue. In the absence of palpable thrombus formation or demonstrable local arterial changes an intimal lesion may still have been present. Only a segmental resection of the artery at this point would definitely have settled the latter possibility. Of significance may be the observation that pain as well as Raynaud's phenomenon during weight carrying could be prevented by preliminary local infiltration with novocain. A possible explanation is that the anesthetic agent interrupted the physiologic action of the sympathetic fibers of the arterial segment thus preventing distal vasoconstriction. On the other hand, without local infiltration the protruding fragment of bone would mechanically stimulate the sympathetic fibers of the arterial segment resulting in vasospasm.

Would the abolition of pain in itself in the aforementioned observations be sufficient to explain the prevention of vasospasm? In other words, can the pain factor in itself lead to Raynaud's phenomenon? In the present case the pain may have been originated by local irritation of the nerve endings in the adventitia of the artery by impingement from the side of the protruding osseous fragment. It may more probably originate from periosteal areas during the expression and depression

of the loose navicular fragment. Irrespective of how the pain originated, the fact remains that pain was a pronounced symptom during weight carrying and, as was expected, disappeared upon local infiltration with novocain.

By closer consideration there appears to exist no need physiologically for a differentiation of vasospasm as caused by mechanical stimulation of adventitial constrictor sympathetic fibers as compared to that resulting from a reflex mechanism on the basis of pain. According to Ranson and Billingsley,[2] "vasoconstrictor fibers are not specific but are identical with those which subserve protopathic sensibility (pain, extremes of temperature)."

If this is so, one may ask why in our case immersion of the hand in cold water did not produce Raynaud's phenomenon? One may postulate differences in the intensity of the stimulus as represented on the one side by the cold factor and on the other by pain produced by weight carrying. It is to be recalled also that the pain associated with weight carrying appeared in a situation with increased local metabolism due to muscle action.

The discussion has been developed to this point as it affords an opportunity to draw parallels from the present simple case to more complex diseases associated with vascular phenomena. The problem of the cervical rib and scalenus anticus syndrome may be considered as an example.

In an occasional case the vascular phenomenon associated with cervical rib or scalenus anticus syndrome may be predominating. Intermittent Raynaud's phenomenon may be present, together with absent or diminished pulsation of the peripheral arteries of the affected arm, leading to trophic disturbances and occasionally to gangrene of the fingers.

An extensive discussion has centered on the physiopathologic disorder of these vascular manifestations. For the sake of simplification two extreme conceptions may be mentioned here. The one represents what may be termed a purely mechanical view, according to which the vascular symptoms derive from simple compression of the subclavian artery by the cervical rib and/or the contractions of the scalenus anticus muscle. According to the other point of view, the vascular changes result from an arterial vasoconstriction due to stimulation of sympathetic fibers of the perivascular or the brachial plexuses. As will be noted this controversy is identical to that around which the discussion has been centered in our case. This is only natural since, by closer considerations, the existing pathologic change in many respects is similar. This similarity consists in an intermittent abnormal relation between a main artery or nerve plexus and the skeletal muscle system.

Granted that the anatomic-pathologic similarity may be striking, still it is not permissible to conclude that the physiopathologic condition would also be identical. However, it may be of some consequence to keep in mind a certain pattern of vascular reactibility taking place under reasonably well controlled and simplified conditions as demonstrated by our case. This pattern is characterized by arterial and arteriolar constriction resulting from mechanical vascular irritation and most probably due to stimulation of the perivascular sympathetic plexus. In the investigative approach to vascular abnormalities in cases of cervical rib such a pattern of vascular reactibility may be worth while keeping in mind.

SUMMARY

A report has been presented of a patient who exhibited a non-united fracture of the navicular bone of twenty-two years' duration, associated with intermittent Raynaud's phenomenon of the corresponding hand. Functional restoration of the hand followed upon operative removal of the projecting movable fragment.

Preoperatively, the Raynaud's phenomenon could be reproduced by weight carrying but not by exposure to cold. Its occurrence

could be prevented by local infiltration of novocain around the loose fragment.

The discussion essentially centers on the mechanism of Raynaud's phenomenon as produced by this relatively simple lesion and under reasonably well controlled clinical conditions. It is concluded that in all probability Raynaud's phenomenon in the present case resulted from stimulation of the perivascular sympathetic plexus leading to vasoconstriction of the distally located arteriolar branches. It is implied that this represents one simple mechanism explaining Raynaud's phenomenon. Others definitely exist. Certain parallels are drawn to the more complex problem of vascular phenomena noted in cases of the cervical rib and scalenus anticus syndrome.

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A. H. Baker reports the case of a four year old patient in whom several diagnostic lumbar punctures were performed. The child developed backache and then difficulty in walking and sitting. The two intervertebral spaces between the second to the fourth lumbar vertebrae later became narrower, according to x-rays, and it is believed that probably the lumbar puncture needle ruptured the annulus and permitted the liquid nucleus pulposus to seep out through this perforation. In adults lumbar puncture is less apt to be followed by such an unusual complication because the nucleus pulposus is more solid in such cases. It is well to keep this possible complication in mind when one is required to perform lumbar punctures in infants and children. (*Richard A. Leonardo*, M. D.)

CYSTIC HEMANGIOMA OF THE SPLEEN

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CAPILLARY and cavernous hemangiomas of the spleen constitute a relatively rare pathologic entity. Most reported cases[1–3] have been from autopsy findings in which an incidence of about 0.1 per cent has been recorded. A few cases have been discovered at operation which has usually been performed for hemorrhage following trauma.[4,5] Hemangioma and lymphangioma are of particular interest in the present report because they may be the underlying basis for cystic changes giving rise to a variety of pathologic changes in the spleen.

In general, it may be stated that cysts of the spleen constitute a pathologic curiosity and are extremely rare in clinical surgery. McClure and Altemeier[6] report 148 splenic cysts in a review of the literature up to 1941. As a cause of splenomegaly, cystic disease is responsible for a very low percentage and, apart from parasitic invasion (e.g., echinococcus disease), only 108 splenic cysts were reported up to 1941 by Watts and Warthen.[7] Deneen[8] states that a review of the Bellevue Hospital statistics from 1904 to 1942 revealed no case of hemorrhagic cysts of the spleen with a similar record at St. Vincent's Hospital for the period 1910 to 1940. McClure and Altemeier[6] add to their report a recent case of a patient operated upon by them at the Henry Ford Hospital. Paul[9] believes that cysts of the spleen are very rare. He states that Andral reported the first case in 1829 and he then reports an operative case in which the spleen presented one large and several smaller cystic areas. In his case there was a history of malaria and in the pathogenesis of this lesion an antecedent malarial infection with trauma appear to be prominent features. Andral's case microscopically presented cyst walls lined by stratified epithelium. The same writer presents a simple classification of splenic cysts as follows: (1) Hydatid cysts of the spleen; (2) multiple serous cysts associated with polycystic disease of the kidneys; (3) single cysts consisting of (*a*) dermoid cysts, (*b*) epidermoid cysts and (*c*) serous and blood cysts devoid of an epithelial lining and (4) Multiple serous cysts.

McClure and Altemeier[6] present a more elaborate classification: (1) true cysts—lined by a specific secretory membrane; (*a*) epithelial consisting of dermal and epidermal; (*b*) endothelial consisting of lymphangioma, hemangioma, polycystic disease and some serous cysts; (*c*) parasitic—lined by protoplasmic matrix containing numerous nuclei making up x-hydatid cysts. (2) false cysts—no specific secretory lining making up (*a*) hemorrhagic; (*b*) serous; (*c*) inflammatory with acute necrosis in infection and chronic tuberculosis; (*d*) degenerative liquefaction of infarcted areas caused by embolism or arterial thrombus.

The subject of the present report appears to have true cysts of endothelial origin. Gradations and changes in the pathologic picture, depending upon the age of the lesion, areas studied, concommitant disease, etc., makes overlapping of classification likely. Sweet[10] like most writers emphasizes the relative paucity of clinical symptoms associated with splenic cysts. Apart from the acute picture of shock and hemorrhage which may occur with rupture, the chief complaints are those referable to the presence of a mass in the hypochondrium; because of its size, this may give rise to varying degrees of discomfort and dysfunction due to pressure on neighboring viscera. Differential diagnosis of splenomegaly ordinarily presents no great diffi-

culty but such conditions as polycystic kidney, retroperitoneal blastoma, mesenteric cysts, etc. must be considered either alone or in association with splenic cysts. The diagnosis of cystic disease *per se* must usually await surgical exploration or necropsy although Sweet and others have pointed out the value of x-ray studies.

Because of the benign nature of the process, splenectomy for splenic cysts gives excellent results. However, the technical problems involved in splenectomy for any splenomegaly present a wide range, and in some cases they may be of such a nature as to preclude successful surgical extirpation of the tumor. These problems have been reviewed by Balfour,[11] Henry[12] Maingot,[13] Behrend[14] and many other surgeons. Recently, Dunphy[15] summarized surgical problems, reviewed surgical anatomy and presented some very important points of great technical significance in splenectomy. We agree with him that splenectomy is often a relatively simple procedure in the case of a small spleen; here, even in the presence of severe trauma and hemorrhage, proper preparation coupled with good anesthesia will ensure a fairly simple and successful operative procedure. However, in long-standing pathologic states (Banti's syndrome, splenic anemias, cirrhosis, etc.) the development of numerous extremely vascular adhesions, the great friability of the spleen and the technical obstacles presented by the size of the organ and its fixation to adjoining viscera (especially the stomach, colon and diaphragm) may combine to render splenectomy extremely hazardous or impossible even for the most experienced and skilled surgeon.

The present case is deemed worthy of presentation not only because of the rarity of the condition but also because of the associated pregnancy, the long history, the striking surgical and pathologic findings and also to reemphasize the surgical difficulties which one may anticipate. We believe it also serves to point out the great value of adequate pre- and postoperative care.

CASE HISTORY

Mrs. B. C. (Hospital No. 7752), a married Negro female, twenty-five years of age, was first admitted to the out-patient service, Herrick Memorial Hospital, April 29, 1946. Her chief complaint was a painful mass in the left hypochondrium of about seven month's duration. Her family history revealed the following: the medical status of her parents was unobtainable; there were several siblings; there was no known history of tuberculosis, cancer or other familial diseases. In regard to the patient's past history she denied that there were any serious illnesses or operations. The patient, on closer questioning, stated that she may have had malaria during her childhood in the South, and also stated that she had "spleen trouble" at the age of thirteen. Catamenia began at the age of 15; her periods were regular every twenty-six days, lasting for three days; there was a moderate flow and no dysmenorrhea. The patient had been married five years. She has two living children; she had one spontaneous miscarriage in 1944. Last menstrual period was on March 13, 1946. She had been troubled by nausea and painful breasts since the onset of her present amenorrhea; she thought she was probably pregnant.

Physical examination revealed a fairly well nourished, young colored woman. Her weight was 143 pounds and blood pressure, 90/60. System findings were unimportant except for abdominopelvic findings. There was a large, irregularly round, firm, slightly tender mass occupying the left hypochondrium. It extended from below the rib cage to the left flank, well below the umbilicus and mesially to the right of the midline. A notched surface was palpable. The liver was not enlarged to percussion in the right hypochondrium and dullness merged with that of the mass on the left side. Abdominopelvic examination showed the uterus to be uniformly enlarged, soft and extending above the symphysis; the mucous membranes of the vault were dusky.

Clinical diagnosis: Early pregnancy; splenic tumor.

X-ray and laboratory findings revealed that a plain film showed extreme enlargement of the spleen; the left costophrenic angle was compressed and there were old, healed tuberculous foci in the left lung; the finding suggested leukemia or other lymphoblastoma. Urinalysis

was within normal limits; repeat blood counts showed mild secondary anemia and moderate leukopenia with normal distribution of leukocytes; fragility tests were within the normal range; platelets, 249,000; blood was group III-B and Rh positive.

Tentative diagnosis: Splenomegaly of unknown etiology, complicated by pregnancy.

The patient was admitted to the hospital July 22, 1946. In view of her generally good condition it was decided to postpone transfusion until surgery and thereafter as a difficult operative course was anticipated. She was operated upon on July 23, 1946, under nitrous-oxide-oxygen ether. Saline was commenced before the abdomen was opened and this was followed with two units of whole blood. A left rectus incision extending from the costal margin to the superior border of the ilium was made. As soon as the abdomen was opened 25 mg. of intocostrin were administered by injection into the perfusion fluid. On opening the peritoneum the spleen presented as a huge cystic mass widely displacing the stomach and the colon. A large knob-like tumor or cyst occupied the lower pole of the spleen and the upper surface was deep under the dome of the diaphragm. There were whitish somewhat raised areas scattered over the remainder of the surface, and the organ was densely adherent to the diaphragm, liver, lateral abdominal wall, transverse colon, stomach and the omentum. These adhesions were very thick, tough and extremely vascular. The splenic tumor was completely immobile until dissection of its attachments was commenced. The gastrosplenic omentum was doubly ligated as high up as possible in serial segments with 2 chromic and carefully cut between ligatures. Because of very dense adhesions in the lesser sac, exposure and control of the major vessels through this approach did not appear feasible. Freeing of adhesions was therefore proceeded with, using sharp and blunt dissection of small zones with secure hemostasis at each step. The peritoneum of the left parietal region above the lienorenal ligament was carefully incised and stripped down with gauze. The right hand was then insinuated between the upper pole of the mass and the diaphragm; here blunt dissection of adhesions had to be accomplished blindly. It was then found possible to push the huge splenic tumor into the wound after which mesial rotation exposed the broad pedicle with the main blood vessels. These were carefully isolated and doubly ligated with No. 4 chromic, a double clamp was placed distally and the pedicle cut through. The pedicle was then transfixed and tied with a third ligature which was then tacked down to the splenic bed. As soon as the spleen was delivered two large hot saline packs were snugly applied to the tumor bed. It was remarkable and very gratifying to note, upon removal of the packs, that the field was dry. The opening in the gastrocolic omentum was repaired with plain 0 catgut. The abdomen was closed in layers with interrupted sutures to muscle, fascia and skin and with four heavy silk retenion sutures. The patient's condition was excellent during the entire procedure. Systolic blood pressure ranged from 120 to 110 and diastolic pressure from 80 to 60 mm. The pulse averaged 90 and respirations 20. The entire procedure took one hour and forty-five minutes. On returning to the ward the patient was given another unit of whole blood, and later that day 1,000 cc. of 10 per cent glucose in saline. The postoperative course was remarkably uneventful. The highest postoperative temperature was 101°F. for one day and the pulse and respiration remained within normal limits. She was taking liquids on the first postoperative day, a light diet on the second day and was ambulatory and on a general diet on the fourth postoperative day. The patient complained of some back pains and pelvic discomfort resembling labor pains on the ninth day; these subsided with sedation. She left the hospital on the twelfth day. The stitches were removed and the wound appeared to be healing nicely per primam. She reported subsequently to the obstetrical service of the out-patient department and pursued an uneventful prenatal course. On November 27, 1946, she was delivered with low forceps of a 7 pound baby girl. Her puerperal status and course were entirely uneventful. The patient has reported subsequently to our Clinic. She was reviewed December 30, 1946, at which time she was feeling fine and had no further complaints.

Pathological report revealed the following: specimen was labelled 'spleen.' It was a large, dusky, cystic mass, measuring about 22 by 15 by 8 cm. On cut sections it appeared very congested and was riddled by variously sized cystic cavities, these varying in size from a few mm. to several cm. in diameter. Microscopic examination of sections from different areas

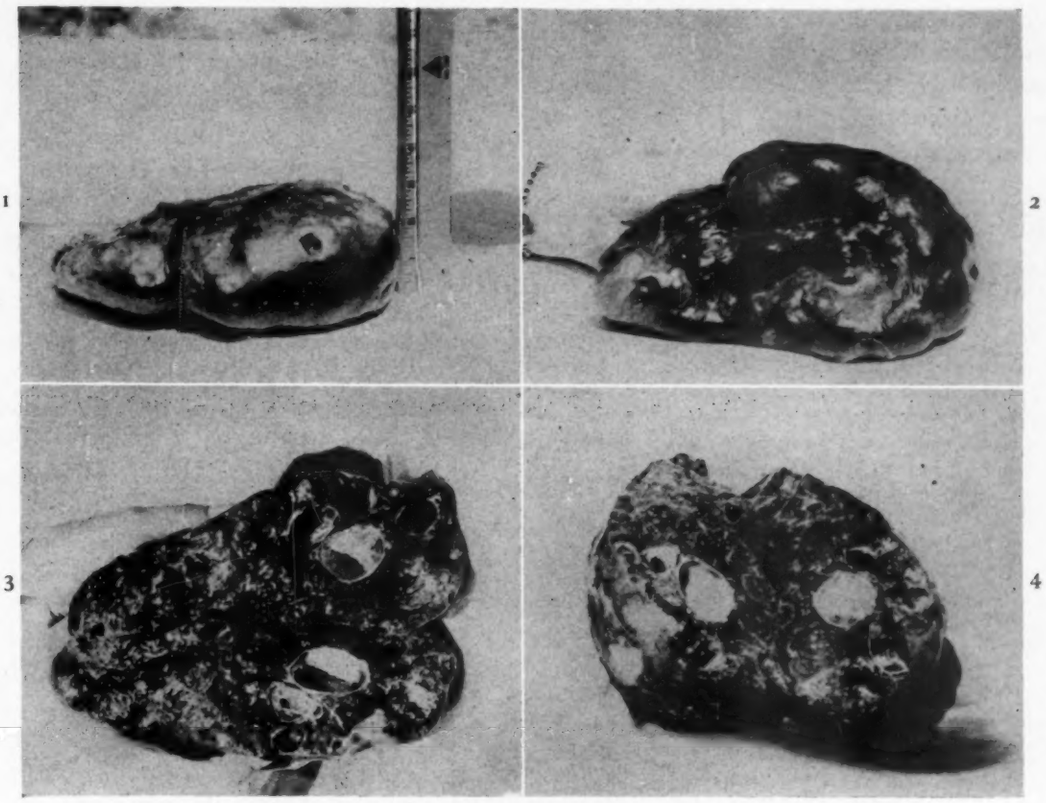

Fig. 1. Superior and lateral surface of splenic tumor; relative size indicated.
Fig. 2. Inferomesial surface showing cystic masses.
Fig. 3. Longitudinal section showing large and small cysts.
Fig. 4. Cross section showing cysts and heavy scarring.

were examined. There was almost complete destruction of the splenic pulp. This had been replaced by broad bands of fibrinous and fibrous tissue which appeared to surround vascular channels lined by endothelium. In some areas a capillary hemangiomatous network was well demonstrated. There was no evidence of malignancy. In other levels the general pattern of a plexiform hemangioma was similarly noted. Wide trabeculae dominated the picture with small islands of blood vessels spreading and compressing the tissue. In the larger spaces a liquefaction necrosis had converted the picture from red blood cells and vessels into a homogeneous acidophilic matrix.

Diagnosis: Cystic degeneration in plexiform hemangioma of the spleen. (Figs. 1, 2, 3 and 4.)

SUMMARY

The clinical history which went back to early childhood is extremely interesting. Also notable is the relative paucity of complaints referable to the growth until it had reached huge proportions. The possibility of a long-standing latent malarial infection may be inferred although no hematologic or pathologic evidence was found in our study. It is interesting to speculate on the possible rôle of trauma in the initiation of hemorrhages and cystic degeneration in an organ which is the site of capillary and cavernous hemangiomas. These conditions would undoubtedly render the spleen unusually susceptible to injury. Again, however, no definite history of trauma was elicited but forgotten injuries may well have occurred repeatedly during the life of the patient. A comparison of the histologic features of our case with those we have had the opportunity to review in the literature leads us to remark on the unusually striking features of hemangioma depicted in this case. The association of a relatively advanced pregnancy as a troublesome complication is of

clinical interest, and the favorable outcome for mother and child indicates to what a degree nature will tolerate a pathologic state and a surgical insult of great magnitude without relinquishing its purpose.

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Cancer of the Islets of Langerhans is a fast-growing tumor, occurring usually in the tail of the pancreas of middle-aged men, with or without hypoglycemia. Unless metastases are discovered, usually in the liver and neighboring lymph nodes, the malignant nature of the growth is likely to escape detection by the surgeon. (*Richard A. Leonardo*, M. D.)

EWING SARCOMA OF THE RIB*

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TUMORS of the chest wall are not rare and, in this group, neoplasms of the costal cage are among the most frequent in occurrence. However, it has been quite unusual to encounter a Ewing sarcoma of a rib, and for that reason this report is being made. The tremendous size of the tumor in the case to be reported constituted an additional cause for this contribution.

A brief and incomplete review of the literature for the past ten years reveals reports of 311 cases of Ewing's sarcoma in five groups of appreciable size.[1–5] Twenty-one of these were designated as primary tumors of the ribs, a total of 6.7 per cent of the five groups of cases. Only one patient was reported to be alive when the various articles were prepared for publication. It is generally accepted that rib tumors of this type are extraordinarily serious with a rapid course and an unfavorable end result.

In general, more than half the cases of Ewing's sarcoma occur before the twentieth year, about 80 per cent before the thirtieth year, and it is rare after the fortieth year of age. More than 70 per cent of the cases are in male patients. Additional tumors of bone soon make their appearance, especially in the base of the skull, pelvis, ribs and vertebrae. Whether these are metastatic tumors or secondary tumors of non-metastatic origin is a point which has been subjected to debate. These usually appear towards the end of the first year or earlier and are often seen first in the skull. In many cases the regional lymph nodes become involved. Pulmonary metastases occur in nearly all cases but they appear late in the course of the disease in contradistinction to osteogenic sarcoma which metastasizes to the lungs earlier and in a more prominent fashion. In late stages nearly the entire marrow system is the seat of tumor tissue with extensive destruction of bone.

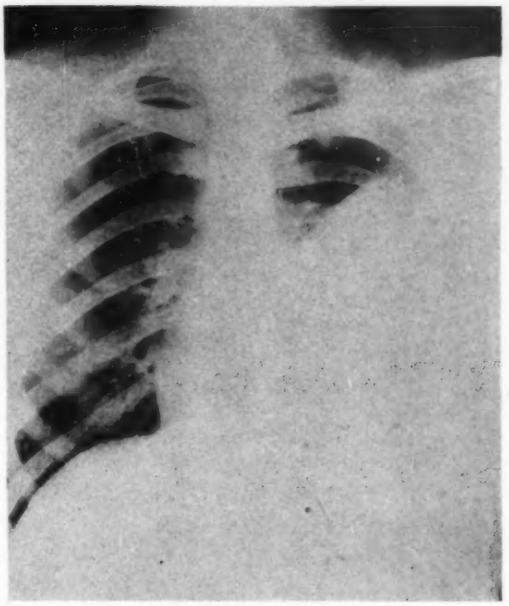

FIG. 1. Chest film revealing the great size of the intrathoracic position of tumor.

Diagnosis is aided by microscopic examination of tumor tissue, the radiographic characteristics of the lesion and by the typically prompt effect of roentgenotherapy upon the tumor. There is, of course, considerable contention as to whether Ewing's sarcoma is an entity, and among those who believe that it does exist, there is disagreement over the cellular origin of the tumor.

Treatment of the original lesion has been accomplished by the use of x-ray therapy, Coley's toxin, curettage, resection or a combination of these methods. The results have been far from satisfactory.

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The opinions or assertions contained herein are the private ones of the writers, and are not to be construed as official or as reflecting the views of the Navy Department or the Naval Service at large.

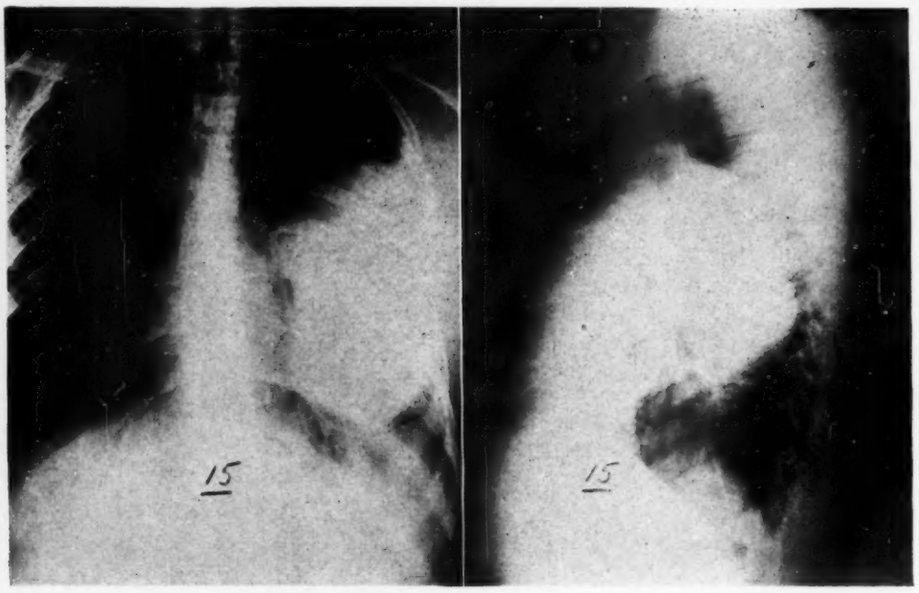

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FIG. 2. Chest film taken five days after institution of x-ray therapy. The effect was prompt and is already quite dramatic.
FIG. 3. Lateral view of chest taken at time Figure 2 was made.

CASE REPORT

A nineteen year old Marine was admitted to the hospital on December 16, 1944, complaining of deformity of the left chest wall and of left-sided pain. The pain had begun one year before and had persisted since onset. The deformity had been noticed by the patient five months before admission and had become progressively more marked.

Physical examination immediately revealed a marked bulging deformity of the left chest wall, chiefly over the anterolateral aspect. The mass was hard and non-tender, poorly defined at the borders and of a large size. Chest x-ray films revealed an enormous intrathoracic extension of the mass. (Fig. 1.)

An aspiration biopsy was performed and a diagnosis of Ewing sarcoma was made. Intensive x-ray therapy was instituted and a total of 5,000 r given, employing 400,000 KV through a single large portal which encompassed the entire extent of the tumor. The radiotherapeutic response was almost immediate after the initiation of treatment as can be seen in Figures 2 and 3. These x-ray films were made only five days after the institution of radiotherapy and a marked reduction in the size of the tumor had occurred. Within ten days, the visible chest wall deformity had disappeared, and at the end of three weeks, the tumor had become much smaller as can be seen in Fig. 4. At this time, considerable destruction of the mid-portion of the left fifth rib could be seen on the x-ray films, indicating that this bone had been the primary site of the neoplasm. The roentgenotherapy had been completed very shortly afterward but no further reduction in size of the tumor could be demonstrated in the ensuing three weeks. Accordingly, the decision was made to resect the remaining tumor mass.

On February 21, 1945, the operation was performed. The major portions of the fourth, fifth and sixth left ribs were removed along with the intercostal structures of the third, fourth, fifth and sixth intercostal spaces. Gross evidence of invasion of the left lung was present. Consequently, about one-third of each of the left pulmonary lobes was resected and the entire specimen was removed *en masse*. The defect in the thoracic cage was necessarily rather large and reconstruction of the boney chest wall by means of tailored ribs of tantalum was considered but the plan was rejected because of the possibility of poor wound healing as a result of the effect of the x-ray treatment upon the soft tissues of the thoracic wall. There had been considerable effect upon the skin. As a result, a minimum of buried sutures were employed in the chest wall closure and retention sutures were inserted to suplement the closure.

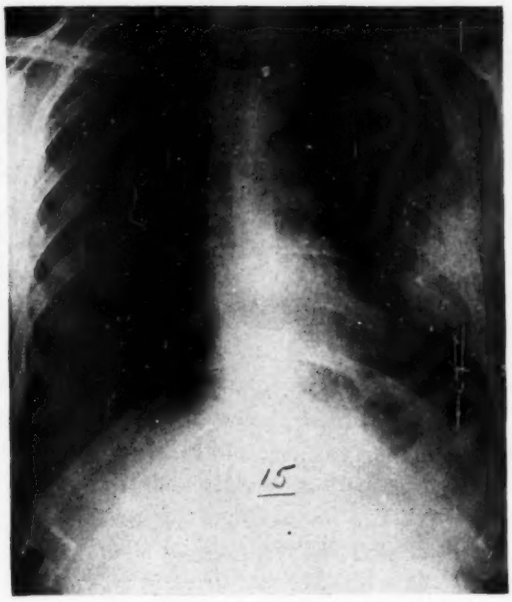

Fig. 4. Chest film taken near the conclusion of x-ray therapy. The reduction in size had taken place over three weeks time but no further effect was noted during the succeeding weeks.

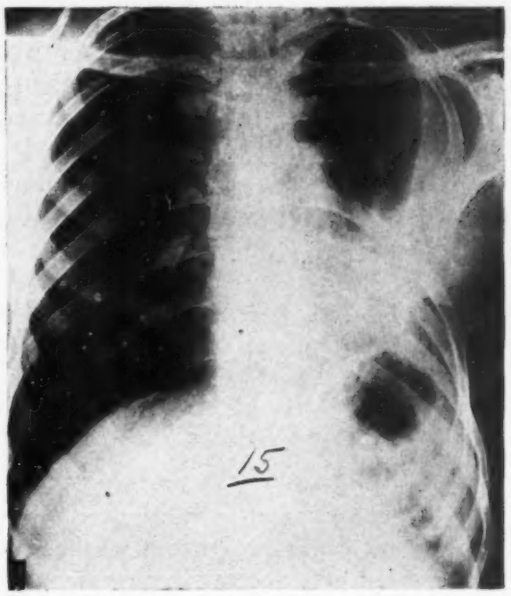

Fig. 5. Chest film made three months after operation. The residual defect in the costal cage is visible. The diaphragm has become elevated. The patient was not short of breath upon exercise.

A catheter was left within the pleural space and brought out through the anterior portion of the wound. This was connected to a water-seal bottle and it was employed to facilitate expansion of the partially resected pulmonary lobes and to permit drainage of blood and fluid from within the thoracic cavity. The catheter was removed on the fourth day. The wound healed promptly except for a small portion of the skin at the anterior limit of the incision which required three and one-half weeks for complete healing. The patient began to get out of bed on the seventh postoperative day but his return to normal was prolonged over a period of one month. During this period there were no evidences of cause for alarm other than a continued slow loss of weight, failure to regain appetite and a discouraging delay in regaining strength and his preoperative state of morale and spirit. However, the improvement in all phases was very rapid after it began and a very satisfactory recovery took place.

A considerable defect in the boney thoracic cage resulted as can be seen in Figure 5, a chest film made three months after operation. There was surprisingly little visible deformity of the chest wall as a result, but the patient was somewhat apprehensive over the loss of the protective function of the missing ribs, particularly anteriorly over the cardiac area. Accordingly, a specially fitted protective device was made which was both comfortable to wear and easily retained in place by two straps. The patient was very well satisfied with the appliance and wore it regularly.

Examination of the surgical specimen revealed that the tumor had arisen in the fifth rib which was partially destroyed, and that it had invaded the attached portions of both pulmonary lobes. Microscopic and gross examination indicated that the invasion had not extended to the line of pulmonary resection. The microscopic picture revealed that the tumor cells had suffered too many destructive changes from the x-ray therapy to permit identification or to allow comparison with the tissue obtained by aspiration biopsy.

Thirteen months after operation the patient was well and without evidence of distant or local recurrence.

SUMMARY

1. A case of Ewing sarcoma of the fifth left rib is described.

2. The tumor had reached astonishingly great size before the patient was received for treatment.

3. The diagnosis was made by aspiration biopsy and supporting evidence is afforded by the rapid and very marked radiosensitivity displayed by the tumor.

4. When a maximum of effect seemed to have been achieved from radiotherapy, a radical resection of the tumor with adjacent chest wall structures and pulmonary tissue was performed.

5. The patient was well thirteen months after operation but the prognosis is very doubtful at best.

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According to S. Annersten, scalenus anticus syndrome is often due to pressure upon the brachial plexus by this muscle, especially when the costotransverse process of the seventh cervical vertebra is unduly large and that often no distinct cervical rib is present. In such cases simple scalenotomy is all that is needed to relieve the patient of his or her symptoms. (*Richard A. Leonardo*, M. D.)

PRIMARY TORSION OF THE OMENTUM*

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PRIMARY torsion of the great omentum is one of the infrequent acute surgical conditions of the abdomen. The literature records but seventy cases. The condition has seldom if ever been correctly diagnosed prior to operation. In nearly all the reported cases the preoperative diagnosis has been acute appendicitis since the clinical symptoms of the two conditions are similar.

Torsion of the omentum means a twisting or rotation of part or all of that structure upon its axis. An ovarian cyst becomes twisted upon its pedicle in the same way.

For many years no attempt was made to distinguish between this type of torsion and that which is commonly observed to occur in hernia or as a result of neoplasms, adhesive bands or inflammatory processes. It remained for Eitel in 1899 to report the first case of primary intra-abdominal torsion occurring independently of these etiologic factors. Later cases were reported by Baldwin and by Syme in 1902 and by Scudder in 1904.

In 1938 Teller and Baskin clarified the subject by presenting the following classification: (1) primary (pure, true, idiopathic) which constitute simple torsion and torsion with herniation of the mass through the omentum itself and (2) secondary which make up hernial; hernio-abdominal; abdominal; pelvic and combined.

In 1946 Altemeier and Holzer reviewed the literature and found sixty-four cases; their six cases from the Cincinnati General Hospital brought the total to seventy recorded cases. Two more cases are added by this report.

CASE REPORTS

CASE 1. G. K., a fifty-two year old, white male, was admitted to the Methodist Hospital January 10, 1939, with a history of gas and bloating for three days accompanied by increasing right abdominal pain. The pain had developed gradually but had remained constant and was without any known cause. It was described as dull and never severe or colic-like. No nausea or vomiting had occurred but there was occasional bitter regurgitation into his mouth.

Examination showed a moderately well nourished middle-aged man obviously in discomfort. His temperature was 100.6°F; his pulse was 100. General physical examination was essentially normal except for the abdominal condition. There was slight rigidity and tenderness in the right upper quadrant; this condition was marked in the right lower quadrant, the maximum point of discomfort being at McBurney's point.

Laboratory examination showed the urinalysis to be essentially negative and the white blood cell count to be 13,800.

Diagnosis of acute appendicitis was made and operation was performed under spinal anesthesia. The abdomen was opened through a pararectus incision, and it was at once noted that the appendix was nearly obliterated and was not the cause of the acute inflammatory symptoms. At the same time free sanguineous fluid was observed and about 8 inches of the great omentum just above the appendix was seen to be strangulated and showed evidence of the start of gangrene. This was the result of primary torsion. Because it was impossible to unravel the omentum, the diseased portion was resected at the pedicle. Appendectomy was performed. A search of the abdomen revealed no evidence of other pathologic disease. The patient made a normal recovery and was discharged on the tenth postoperative day.

Pathologic examination showed an omental mass approximately 8 by 6 inches, the blood vessels of which were thrombosed; there was hemorrhagic extravasation, necrosis and gangrene.

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For the past fifteen years I have preferred the pararectus to the McBurney incision because it permits better exposure of the abdomen for just such unusual conditions as this or for a difficult appendectomy.

Case II. J. S., a white male forty-one years of age, was admitted to the Methodist Hospital December 3, 1942, with a diagnosis of acute appendicitis. For thirty-six hours he had complained of a steady, dull pain in the lower right side. There was no diarrhea or constipation but he was nauseated and had vomited once. He complained of marked tenderness on pressure. There were no urinary complaints and no history of a chill.

The temperature was 101°F., the white blood count was 17,240 and the urinalysis was negative.

Physical examination was negative except for moderate rigidity and tenderness in the right lower quadrant. The patient's discomfort seemed out of proportion to the clinical findings, but the high blood count and polymorphonuclear count of 72 suggested a probable gangrenous appendix.

Immediate operation was advised. Under spinal anesthesia a pararectus incision was made and the appendix located. It was at once evident that this was not the source of the trouble; further search revealed a mass in the right upper quadrant. The incision was extended upward, and the mass was revealed to be a primary torsion of the omentum. The latter was necrotic at the lower 3 inches and showed evidence of marked inflammatory reaction and hemorrhage. The diseased tissue was resected at the pedicle, the appendix was removed and, further examination revealing no other abnormality, the abdomen was closed. Convalescence was normal and the patient was discharged seven days later.

Pathologic examination showed a necrotic piece of omental tissue approximately 6 by 4 inches with a marked torsion at the neck. The vessels were thrombotic and the tissue showed evidence of the start of gangrene.

ETIOLOGY

The etiology of primary torsion of the omentum is unknown. Nearly all the reported cases occurred on the right side, and since this side of the omentum is larger and more mobile than the left side it would be more apt to be involved by exaggeration of the normal movement of the omentum. Some observers, such as Teller and Baskin, thought that obesity might be a factor but neither of the author's cases occurred in obese persons. Others have reported trauma as a possible cause. Payr believed that hemodynamic forces might be important and Sellheim believed that rotary movement of the body was a factor. These are all hypotheses and the real cause remains a mystery.

DIAGNOSIS

About the closest that one may expect to come in diagnosing this condition is to state that the patient has an acute surgical condition of the abdomen and should have an immediate exploration. The writer is fairly confident that no one could have correctly diagnosed the two patients he operated upon although one might have hazarded a guess. When one stops to consider that about 300,000 appendectomies are performed yearly in this country and that only seventy-two cases of primary omental torsion have been reported, one sees what a long chance one would be taking in naming this condition as a preoperative diagnosis. The symptoms are nearly identical as are the physical findings. It is true that in the second patient I undoubtedly was able to palpate the rolled-up and twisted omentum. I assumed it was just that but I had expected it to surround a gangrenous appendix.

Altemeier and Holzer believe that the progression of symptoms is usually less rapid than in appendicitis. This may be true but it has been the author's experience that in appendicitis the progression of symptoms may be rapid or slow, depending upon the resistance of the individual, the virulence of the organisms and the manner of treatment the patient receives. As these authors so well state, however, it should be emphasized that no practical importance is attached to a preoperative diagnosis of

omental torsion since the similarity of the signs and symptoms to those of acute appendicitis make surgical exploration of the abdomen mandatory.

There are a few important factors about this condition that the writer believes should be emphasized. Because of the rarity of the condition, there is the possibility that it may be overlooked and only a non-responsible appendix removed. The condition is one calling for immediate surgery to prevent the process from becoming extensive and so endangering the patient's life. A thorough resection of the diseased omentum should be performed with careful ligation of the blood vessels. When surprised on opening the abdomen not to find the appendix the cause, bear in mind omental torsion, regional enteritis, Meckel's diverticulum not to mention a host of other conditions. In the acute surgical condition of the abdomen there are surprises that always keep the experienced surgeon in a humble mood.

CONCLUSIONS

Two cases of primary omental torsion are added to the seventy previously reported cases in the literature.

1. The etiology of this condition remains unknown.

2. Primary is to be differentiated from secondary omental torsion. The latter condition is observed to occur commonly in hernias.

3. There are no distinguishing clinical symptoms in this condition.

4. Primary omental torsion closely simulates acute appendicitis and calls for immediate exploratory laparotomy.

5. Only one death resulting from operation is recorded in the literature.

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SULFATHALIDINE IN LOW POSTOPERATIVE FISTULAS OF THE ILEUM*

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USE of sulfasuxidine and sulfathalidine† in the treatment of surgery of the large bowel has been definitely established. Sulfathalidine or phthalylsulfathiazole, the most recently introduced agent in colonic surgery, was introduced by Poth and Ross[1] in 1943. Poth and Knotts[2] noted that diarrhea and purgatives adversely affected the use of sulfasuxidine in surgery of the colon. The purpose of this paper is to show that sulfathalidine is helpful and of value in postoperative fistulas involving the lower ileum.

Poth[3] and Angelo[4] in their articles summarize the advantages of sulfathalidine as follows: (1) It has a more rapid action on Bacillus coli and the vegetative forms of clostridia; (2) the feces become soft and not fluid; (3) it is effective in diarrhea; (4) no toxicity has been reported as yet; (5) it is twice as potent as sulfasuxidine and so the dose need only be one-half as much.

Poth, Ross and Fernandez[5] working on dogs showed that when sulfathalidine was used preoperatively and postoperatively, the inflammation, resulting from spillage of intestinal contents through the suture line in open anastomosis, subsides and the tissues undergo orderly repair and healing. Whereas 43 per cent of the controls died from generalized peritonitis, none died when the drug was used.

Poth states that in the calculation of the dosage it should not be the weight of the patient, but the inner surface of the bowel area and the rate at which this drug passes over this that determines the quantity necessary to effect a desired action.

The following are two case reports of intestinal fistulas that resulted from intestinal resection. Penicillin and sulfadiazine were used without result. One of the patients had in addition a disruption of the entire wound with multiple fistulas. These patients were put on a dosage of 2 Gm. of sulfathalidine every four hours although Angelo in his series of patients believes that only 5 Gm. in divided doses are necessary.

CASE REPORTS

CASE 1. L. B. W., aged thirty-eight, was admitted on August 7, 1946, with a two-day history of nausea and vomiting. This was accompanied with intense colicky pain. A previous operation was done on the gynecological service, March 8, 1946. At that time a right salpino-oophorectomy and left cornual resection was done.

On examination an acutely ill, colored female was seen. All essential findings were localized to the abdomen. The latter was moderately distended with a mass in the lower suprapubic area. The white count was 31,350 with 94 per cent polymorphonuclear. The red blood count was 3,720,000 with 100 per cent hemoglobin and a sedimentation rate of 18 mm. in twenty minutes. The urinalysis was negative.

The patient was taken to the operating room at which time 2 feet of gangrenous bowel (ileum) was removed. An end-to-end anastomosis of ileum over a Murphy button was done because of the extremely toxic condition of the patient; this type of procedure was used because it allowed the surgeon to complete the operation in the shortest possible time.

Postoperatively the patient was put on penicillin, Miller-Abbott tube, Wangensteen drainage, intravenous fluids, plasma and blood. The course was extremely stormy; the patient became delirious and toxic. On August 14, 1946, the patient had a passage of flatus and feces. On August 17, 1946, the patient developed an intestinal fistula. Sutures were removed; the fecal drainage was profuse.

On August 19, 1946, sulfathalidine was started. An initial dose of six Gm. and 3 Gm. four times a day was used. On August 24, 1946,

† The sulfathalidine used was supplied by the courtesy of Sharp and Dohme.

* From the Surgical Service of the Harlem Hospital, New York, N. Y. Read before the Harlem Surgical Society, October 2, 1946.

the fecal drainage was markedly reduced. On August 26, 1946, there was only very slight fecal drainage. On August 29, 1946, there was no fecal material but a slight purulent discharge. It is to be noted that the temperature until the start of sulfathalidine ranged between 101 and 102°F.; since then the temperature has remained flat.

Laboratory findings revealed the following: On August 8, 1946, the white blood count was 31,350 with 94 per cent polymorphonuclears; the red blood count was 3,720,000 with 100 per cent hemoglobin; the sedimentation rate was 18 mm. in twenty minutes; creatinine was 1.3; urea nitrogen was 15.0 and glucose was 76.0. On August 14, 1946, hematocrit was 40.0; sulfa level, trace; total proteins, 5.87; albumin, 2.99; globulin, 2.88. On August 19, 1946, hematocrit was 47.5; albumin, 4.14; globulin, 2.39; total proteins, 6.53; Wassermann, negative; urine, negative. On August 21, 1946, the white blood count was 11,200 with 74 per cent polymorphonuclears. The red blood count was 3,210,000 with 74 per cent hemoglobin. On August 28, 1946 the white blood count was 10,500 with 82 per cent polymorphonuclears and the red blood count was 3,460,000 with 70 per cent hemoglobin.

Case II. M. G., aged thirty-one, was admitted on June 11, 1946, with a nine-hour history of right lower quadrant pain associated with nausea and vomiting. Her last bowel movement was on the morning of admission via an enema. Neither a previous history of pain nor a previous operation could be ascertained.

On physical examination a young, colored female in good condition was seen. All essential findings were in the abdomen. There a mass was noted extending from the right inguinal region to the umbilicus. This mass was cystic, round, well demarcated and non-tender. On vaginal examination the same cystic mass was in the right fornix. The impression then was an ovarian cyst. Ten hours later the patient complained of pain in the right side. She was cold, clammy and in shock. The impression was a twisted ovarian cyst. On operation about 6 feet of gut was found extending proximally 4 inches from the ileocecal valve. The operation was an ileotransverse colostomy end-to-side, done with a Furniss clamp.

Postoperatively the patient was put on Miller-Abbott tube, Wangensteen suction and was given plasma, blood, intravenous fluids and penicillin. The patient was delirious the first few days. On June 20th, an infection of the lower angle of the wound resulted. The Michel clips were removed and a Penrose drain inserted. On June 28th, the entire wound disrupted down to the peritoneum and a profuse fecal discharge was noted. The patient was continued on penicillin, but the intestinal fistula persisted. The question of side tracking the fistula arose. Meanwhile the patient was put on sulfathalidine, 6 Gm. for the first dose and 2 Gm. every four hours. Seven days later no fecal drainage was noted. The wound granulated nicely.

Laboratory findings revealed the following: on June 13, 1946, creatinine was 1.3; urea nitrogen, 8.0; sugar, 100; Kahn, 4 plus; urine, negative; white blood count, 11,850 with 87 per cent polymorphonuclears and red blood count, 4,300,000 with 78 per cent hemoglobin. On July 29, 1946, creatinine was 1.3; urea nitrogen, 9.0; glucose, 75.0; chlorides, 472; albumin, 4.14; globulin, 2.97; total proteins, 7.11.

In these two cases the clinical condition of the patients was such that it was our opinion and the opinion of several experienced surgeons that further operative intervention was indicated to close the existing fistulas, but after we put the patients on sulfathalidine they closed without further operative procedures.

CONCLUSIONS

1. Sulfathalidine should be tried in all cases of low postoperative fistulas of the ileum before any operative procedure is attempted.

2. Sulfathalidine's antiseptic action is apparently effective in the lower ileum as well as in the colon.

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MESENTERIC THROMBOSIS OF LOWER ILEUM FOLLOWING RESECTION OF THE SIGMOID COLON FOR CARCINOMA

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IN the case to be described resection of the sigmoid for obstructing carcinoma was followed by mesenteric thrombosis in the terminal ileum. Following successful resection of 14 inches of ileum, obstruction due to adhesions developed, necessitating a third laparotomy. Recovery ensued. We think this sequence of events is interesting enough to warrant a brief report:

CASE REPORT

Mr. H. M., aged fifty-eight, was first seen June 6, 1947. He had bleeding per rectum for four or five months which was apparently no worse in the past week than it was before. For the past month he had passed more gas than usual and for this length of time there had been frequent, small bowel movements and some "gurgling" in the abdomen. Also during the past month there had been pain in the left side of the abdomen, intermittent in character and not severe. This was always better following a bowel movement.

Examination revealed a well developed man, rather small of stature. General examination was negative except for some slight tenderness in the left side of the abdomen and some suggestion of gas. X-ray showed a definite filling defect in the mid-sigmoid. Preoperative films also showed a rounded, soft tissue mass in the region of the bladder which the roentgenologist thought might be a distended bladder.

After careful preoperative cleansing, oral sulfasuxidine, etc., operation was performed on June 16, 1947. Resection of carcinoma of the sigmoid and aseptic end-to-end anastomosis was done. Exploration was carried out through the left mid-rectus incision which was about 4½ inches long. The bladder was tremendously dilated. A catheter was passed into the bladder and fully 2,000 cc. of urine were evacuated. The mass was in the mid-sigmoid about 4 inches above the lower peritoneal reflection. It was about 2 inches long, seemed to encircle the bowel and there were a few dense masses in the mesentery immediately adjacent to the bowel. The remainder of the mesentery was free and the region of the growth itself in the sigmoid was not attached anywhere.

Exploration of the liver revealed two shotty, pea-sized nodules on the lower surface of the liver which seemed to be elevated. To palpation they seemed like typical metastatic nodules, but they were not visualized because of the exposure on the left side. There were no other masses in the liver and no other masses along the posterior lymphatics. Accordingly, resection of the tumor was done with immediate aseptic end-to-end anastomosis. The tumor was mobilized and the peritoneum on the outer and medial surfaces was incised. The ureter was easily seen. The spermatic vessels were seen and the growth was removed down to the superior hemorrhoidal vessels. This was a very large trunk and it was dissected cleanly. The bowel was divided about 1 inch below the growth and about 3 inches above the growth. The division was made over a Wolfson clamp and the bowel cut through and then cauterized. Immediate end-to-end suture was made over two crushing clamps, using other surface interrupted silk and inner chromic sutures. At the conclusion there seemed to be a very good anastomosis and the junction admitted the end of the thumb easily. The peritoneum was then sutured together both on the mesial side and on the lateral side. Apparently hemostasis was satisfactory. The wound was closed.

Pathological diagnosis: adenocarcinoma of sigmoid; metastases to adjacent lymph nodes and to epiploic fat.

Convalescence was fairly satisfactory. There was some ileus for the first few days but on the sixth postoperative day the bowels moved normally and continued to do so. The patient had to be catheterized regularly after the operation. The urologist (Dr. W. K. Rexford) diagnosed a mild cord bladder, symptomati-

cally present before the operation (x-ray appearance of enlarged bladder and dilated bladder at the time of the operation) and on June 27, 1947, a transurethral punch operation was performed. "There was some obstruction of the bladder neck. Three or four pieces of tissue were removed from the floor of the bladder neck. Bilateral vas ligation was done." There was very little reaction from this operation but suddenly on June 29, 1947, the patient began to have some diarrhea.

On June 30, 1947, he was uncomfortable with recurrent cramps. There was some distention toward evening, chiefly in the right lower quadrant where a vague sensation of a mass could be felt in the right rectus region. There was no spasm or rigidity of the abdominal wall. White blood cells were 12,000; hemoglobin, 14 Gm. = 91 per cent; red blood cells, 5.07. The temperature had gone up to 100.4°F. after the transurethral punch operation but had been 99.2 to 98.6°F. since then. There was also some evidence of shock, in that there was cyanosis, sweating and coldness of the extremities. Blood pressure was maintained satisfactorily. Preoperative diagnosis of recurrent intestinal obstruction was made and the patient was taken to the operating room on the evening of June 30, 1947.

A long McBurney incision was made just above the right crest of the ilium. On entering the abdomen some dark colored serous fluid was aspirated. There was a loop of very dark colored small bowel apparent toward the midportion of the abdomen from the incision. The incision was prolonged downward and upward, partially cutting the muscular fibers of the abdominal wall. It was then seen that there was a loop of bowel (small bowel) which was totally gangrenous. It was about 16 to 18 inches in length and ended sharply. At a sharp demarkation point of the bowel both proximally and distally the bowel was viable. The mesentery itself did not look too unusual. There was no marked dilatation either above or below this area and the large bowel was visible in the depth of the wound; this was not dilated. Evidently the condition was a localized mesenteric thrombosis. As far as exploration would allow no larger extent than has just been described was involved. The involved bowel was tense, leathery, engorged and practically black.

Ochsner clamps were applied through the viable bowel, both above and below the gangrenous portion. The mesentery of the gangrenous portion was excised between clamps. An aseptic anastomosis was then done over the two Ochsner clamps, interrupted silk for the outer layers and running No. 00 chromic for the inner layer. When the anastomosis was made, a good lumen was palpated through the anastomotic line and the anastomosis itself looked clean. A few sutures tacked the mesentery together and the abdomen was closed without drainage. The condition of the patient at the end of the operation was much better than it was before. The shock had disappeared, the extremities were warm and the pulse was quiet. He was placed on dicumarol therapy for one week.

Following this, there was still some trouble with abdominal distention; although his bowels moved five days postoperatively and he continued to pass gas and liquid stools, his abdomen was not normal. The Levine tube was replaced by a Cantor tube on the fifth postoperative day. Its progress down the intestinal loops was slow. Gas and feces would follow an enema and flatus would pass frequently, but large amounts of gas and small intestinal contents were always brought up with the Cantor tube. He had persistent extrarenal azotemia, i.e., he was drowsy and the blood nitrogen hovered around 70 to 50 and would not get lower. Urinary output was well maintained (1,000 to 1,500); chlorides, blood proteins, hemoglobin, red blood and white blood cells were normal.

After two weeks went by it was evident that although we could maintain him in electrolyte balance and his bowels moved, there was some partial obstruction of the small bowel which would not allow normal passage. If the long intestinal tube was clamped for two to three hours, he was uncomfortable and distended. If clamped more than three hours, he was tense and in pain. As soon as the clamp was opened he became rapidly deflated in two to three minutes with passage of considerable gas and liquid through the tube.

The patient was re-explored July 18, 1947. Exploration was done through a right midrectus incision. The bowel which presented was not dilated but the wall was thickened. The large Cantor tube was easily palpable in the lumen; it had completely deflated the bowel. The bag containing mercury was found about

3 inches above a tightly adherent area between the small bowel and the posterior peritoneum. The bag was about 3 inches long and about 1½ inches in diameter and had apparently absorbed some air from the bowel. The constricted and obstructed area was in the lower ileum. Here the small bowel was densely adherent to a tag of posterior peritoneum in the region of the promontory of the sacrum. This was dissected free, the bowel serosa repaired and the raw area which was left in the posterior peritoneal region tacked together. About 6 inches below this was the site of the end-to-end anastomosis. This was also caught tightly in a constricting band between the small bowel and the mesentery. When this was liberated, the end-to-end anastomosis was seen to be satisfactory. The finger easily invaginated in it and there was no obstruction. The raw areas here were repaired as well as possible. This area was about 8 or 10 inches from the ileocecal valve. A small piece of oxycel gauze was put against the raw area in the posterior peritoneum where the bowel had been adherent.

There was no exudate in the abdominal cavity and no discoloration or lack of luster of the serosa of the small bowel. The remainder of the abdomen was not explored. There was definitely no further advancement of the mesenteric thrombosis process so no exploration was done for any sign of further extension of the malignancy. The wound was closed in the usual fashion. The condition of the patient at the end was good.

From then on the patient's convalescence was satisfactory. He was discharged from the hospital July 30, 1947. His bowels were moving normally. He subsequently had a transurethral prostatectomy (December, 1947). When last seen on April 1, 1948, he was gaining weight, going to business daily, his hemoglobin was 88 per cent and his bowels moved without discomfort. He urinated easily every two to three hours, felt alert and well and had no complaints.

Whittaker and Pemberton[1] analyzed sixty cases of mesenteric thrombosis at the Mayo Clinic in 1938, and Laufman and Scheinberg[2] carefully studied forty-two cases in Michael Reese Hospital, Chicago in 1942. Their mortalities were 90 to 95 per cent. Thirty to forty per cent of the cases in each series followed operations; the list comprised chiefly splenectomy, stomach, biliary, pancreas, colon and gangrenous appendix operations. In Pemberton's series in only three cases, which followed operation was reoperation attempted. Only one survived; a patient who had a mesenteric thrombosis following hysterectomy. Sixteen of his other patients were operated upon for primary mesenteric thrombosis with only two survivals. Nine of Laufman's forty-two patients were operated upon with only three survivals.

As Laufman and Scheinberg pointed out there are several reasons for the high mortality in mesenteric thrombosis. The patient is very often moribund at the time it is recognized; auricular fibrillation, circulatory failure, etc. are common. The thrombotic process may involve the entire superior mesenteric vessels immediately, death ensuing before gangrene develops. The process of thrombosis frequently continues to extend and propagate itself after operation. Murray[5] believes that this is the principal reason for high mortality. Finally, there is termination in gangrene and peritonitis.

By and large the recovered patients have been those who have undergone resections of relatively small segments of intestine. Brown,[3] in a review of the literature, estimated that removal of up to 3 feet of small bowel carried a hopeful prognosis; any more extensive involvement "sharply increases the mortality."* In our case the resected specimen measured 14 inches, well within this limitation. Laufman and Scheinberg suggested that larger quantities of blood than the usual 500 cc. are required in these cases and that perhaps more frequent use of larger quantities of blood (up to 2,500 cc.) would lower the mortality. Murray gave heparin postoperatively to six patients with mesenteric thromboses in

* Madding and McIntire[4] recently reported a case in which the entire small bowel except the upper 4½ feet of the jejunum and the entire ascending colon to the hepatic flexure was involved. This entire length of bowel was removed and jejunotransverse colostomy done. The patient recovered.

whom 1½ to 7 feet of bowel was removed. He believed that thereby propagation of the thrombus would be stopped. Four of these patients were living and well one year later, and it was the judgement of the operating surgeons that "none of these would have recovered." Much has been written recently about the usefulness of paravertebral novocain block in relieving the arterial spasm which accompanies venous thrombosis of the extremities. Edgar Allen[7] is critical of its value in these cases either for relieving spasm, except occasionally, or for stopping propagation of the venous thrombosis. Tetraethyl ammonium chloride has a similar paralyzing effect on the sympathetic and parasympathetic ganglia. A generalized body effect lasting six to eight hours can be obtained with an intramuscular injection. We have seen no series of cases reported since 1942 in which the previously mentioned advances in surgical care have been evaluated.

Regarding etiology, it is known that mesenteric thrombosis is prone to follow splenectomy when factors conducive to thrombosis are present, and it also follows abdominal suppuration or operations in which soiling has taken place. In our patient it followed colon resection and aseptic end-to-end anastomosis. Recent reports, although favoring aseptic end-to-end anastomosis when feasible, do not show statistically that there is any increased safety or lessened morbidity over so-called open anastomosis. In this latter technic there is bound to be some spillage, but the increased accuracy of suturing and the viability of a non-crushed bowel edge are compensating advantages. Whether the ileus our patient had for five days postoperatively, which was a bit more than usual, indicated slight peritoneal soiling is doubtful. He had no signs of cardiac trouble either before or after operation.

Regarding diagnosis, there is no uniformity to the pain in mesenteric thrombosis. "The pain is a true visceral pain, not associated with muscular spasm or tenderness of the abdominal wall. It differs from simple vascular pain only in its constancy."[6] Until or unless gangrene or peritonitis develops, there is little abdominal wall spasm.

Most observers believe that they cannot accurately tell venous from arterial mesenteric thrombosis except in general terms; venous occlusion tends to develop more slowly, pain and collapse are less. If a small segment is involved or venous thrombosis develops slowly, the pain is less. If the main artery is suddenly occluded, pain is agonizing, severe and there is considerable shock and collapse. Pain usually overshadows vomiting, as a differential point from intestinal obstruction. The pain of our patient was more of a crampy discomfort, constant but not severe. There was a vague mass. Distention was present but abdominal wall spasm was just beginning.

Regarding the operative indications in our patient, the second operation, at which mesenteric thrombosis was found, was done without delay. There was a tender mass and signs of beginning peripheral collapse. By contrast the third obstruction was operated upon only after eighteen days of careful observation and meticulous preparation. This delay paid dividends. It took more than one week for the long intestinal tube to find its way down the length of the small bowel, but since the patient's fluid, electrolyte balance, etc. were maintained there was no hurry. At the third abdominal operation the small bowel was completely deflated and although thickened it could be handled, sutured and adhesions dissected as if it were normal and not obstructed. Recent reports are bearing this out. The lowest mortalities are obtained when operation is not undertaken until the bowel has been completely deflated provided, of course, that strangulation can be ruled out.

SUMMARY

A case is described in which three successive operations for intestinal obstruc-

tion were done; resection and end-to-end sigmoid anastomosis for cancer, resection of the ileum for mesenteric thrombosis, and dissection and liberation of constricting adhesions for simple postoperative obstruction. The indications and clinical course especially as they pertain to mesenteric thrombosis are briefly discussed.

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Macalpine reviewed nineteen cases of papilloma of the renal pelvis and found that diagnosis was facilitated by finding tumor cells in the urine and a growth projecting into the ureter with a characteristic filling defect. Clinically, the condition often simulates hypernephroma. Removal of the kidney and of the whole of the ureter flush with the bladder is the recommended treatment. If the renal pelvis is opened, papillomatous tissue may escape and become transplanted and grow, causing future complications. In four of the reviewed cases this occurred and such transplants assume malignant characteristics. Early diagnosis and complete ureteronephrectomy are therefore necessary if one is to obtain satisfactory results. Hematuria is often an initial symptom in these cases of papillomatous disease. (*Richard A. Leonardo*, M.D.)

New Instrument

SUBPERIOSTEAL PERICOSTAL BAND FOR THORACIC CAGE APPROXIMATION

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THE problem of closing the chest following intrathoracic surgery is often associated with many difficulties. In former years the method of closure went through the same periods of trial and error as confronted the abdominal surgeon. Thus, the thoracic surgeon has passed through the era of catgut, silk and, finally, stainless steel wire. The methods have been far from ideal. These ligatures have been applied extraperiosteally without considering the intercostal nerves. For this reason many thoracic surgeons have abandoned this method and are suturing only the periosteal bed. Some men employ pericostal sutures; however, they place them beneath the periosteum. This method[1] was suggested by the author and has the advantage of eliminating postoperative pain in the chest wall due to compression of the sensitive periosteum and intercostal nerves by the encircling sutures.

1

2

FIG. 1. Photograph illustrating removal of segment of periosteum.
FIG. 2. Technic for applying band.

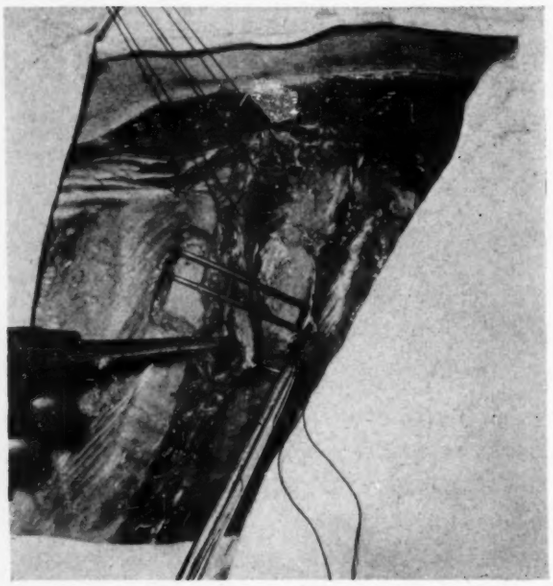

Fig. 3. Band *in situ* with interrupted silk closure of periosteal bed.

As a new method of closing the thoracic cage which is an improvement over former methods, including the procedure already mentioned, the author has employed a metal band. Its use not only successfully prevents postoperative pain but, in addition, has many other advantages. These advantages are: (1) It is very simple to apply. (2) It diminishes the time required for emergency closing of the chest cavity. (3) The use of this approximator necessitates the removal of only small segments of the periosteum. (4) It is a rib approximator which obviates the necessity of employing special instruments and additional assistants to hold the ribs in approximation for suturing. (5) Its steel construction results in a strong chest wall which cannot separate and thus prevents the possibility of wound disruption. (6) The resulting chest wall enables the patient, immediately following the operation, not only to breathe normally but also to turn from side to side and cough if necessary without fear of wound dehiscence and thereby the natural expulsion reflex is preserved. (7) When employed properly, the band does not impinge upon the intercostal nerve and prevents irritation of the sensitive periosteum.

This metal band is constructed of stainless steel and made in varying lengths which have been numbered from one to

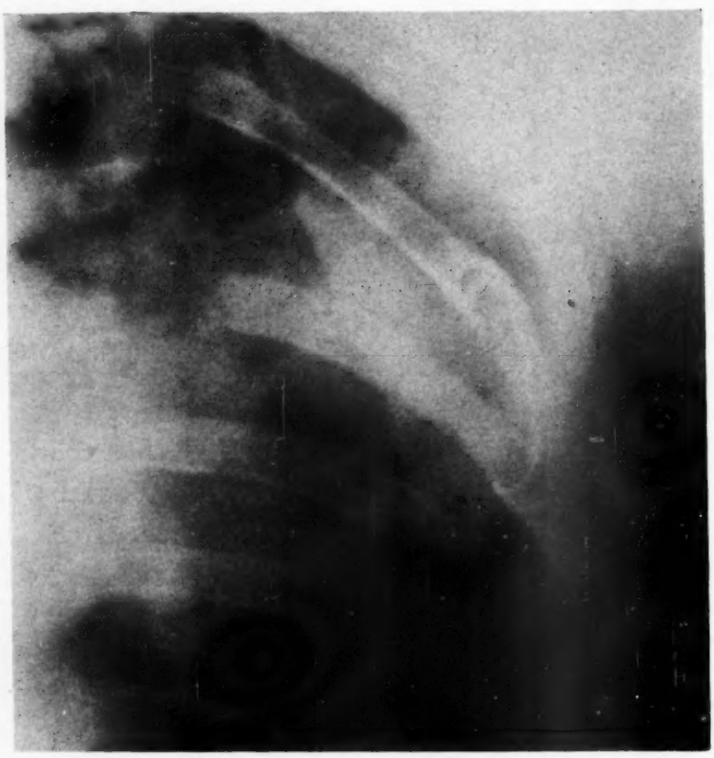

Fig. 4. Postoperative x-ray illustrating the clip.

five.* Number one corresponds to 3 cm. and number five corresponds to 5 cm. All are of uniform width. These clips are applied manually while the assistant holds the ribs to be approximated with rake retractors. When this has been accomplished, the surgeon then approximates the soft tissues of the chest wall. (Figs. 1 to 3.)

This method of closing the chest wall has been employed on many occasions with great ease. Following this procedure, patients have no discomfort. It has never been employed in children because the thoracic cage has not reached its maximum growth and the presence of the clip may influence proper anatomic and physiologic growth. In the event that it becomes necessary to remove the clip, this may be done under local novocaine anesthesia with minimal discomfort or distress to the patient. However, the occasion has never presented itself which necessitated the removal of the metal band.

* Manufactured by The George P. Pilling and Son Co., Philadelphia, Pa.

CONCLUSIONS

A new method is presented for closing the operative wound following intrathoracic surgery. This method employs a metal band of stainless steel of varying lengths. Usually one or two clips are sufficient for approximating and maintaining the ribs. The advantages of this method have been enumerated. It is realized that some disadvantages will arise in the future. However, up to the present time the metal clip has proven to be most satisfactory.

The author wishes to thank Dr. Richard H. Overholt for his cooperation in the use of this clip on his service at the New England Deaconess Hospital, Boston, Massachusetts.

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**Sebrell, W. H., Jr., et al: J. Pediat. 22:494-507, April, 1943.*

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> At the AMA Section on Pediatrics, an outstanding paper on whooping cough serums concluded with this statement: "Our results suggest that human hyperimmune serum or globulin should be used in the treatment of all infants who are seriously ill with whooping cough." *1.*
>
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1. Kohn, Fischer, et al., Am. Jour. Dis. Child. Sept., 1947

2. Brainerd, Henry, Jour. Ped. Jan., 1948

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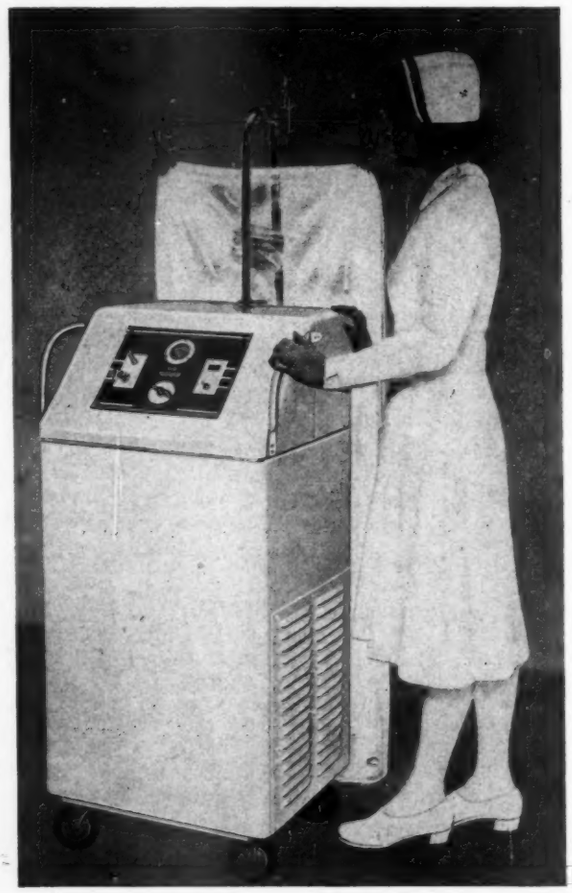
(*continued from page 20*)

THE American Radium Society will meet at the Stevens Hotel, Chicago, June 20–22, 1948. The following preliminary program has been announced:

Experimental Comparison of Gamma and X-rays in Serial Biopsies by Constance Wood; Intra-oral Cancer by Elis Berven; Cancer of Pharynx and Larynx by Francois Baclesse; Cancer of the Uterus by Juliette Baud; Cancer of the Penis by Sir Stanford Cade and B. W. Windeyer; Carcinoma of the Antrum by Sir Stanford Cade and B. W. Windeyer; Preliminary Report upon the Use of Needles Containing Cobalt 60 by William G. Meyers; Roentgentherapy of Advanced Cancer of the Cervic by Francois Baclesse; Contact X-ray Therapy of Cancer of the Rectum by Paul Lamarque; Treatment of Metastatic Cancer of the Breast by J. R. Freid, H. Goldberg and A. Herrman; Cancer of the Ear by C. L. Martin and J. A. Martin; Radiumtherapy of Advanced Salivary Gland Tumors of the Palate by Max Cutler; and Radiumtherapy of Bartholin Gland Cancer by H. H. Bowing. The general discussion will be opened by Dr. Frank E. Adair.

The Janeway Lecture on The Achievement of Radium in the Fight Against Cancer will be delivered by Sir Stanford Cade, and at the banquet an address on Fifty Years of Radium will be made by Edith H. Quimby. In addition panel discussions will be held on the end results of the treatment of cancer of the tongue, should radiotherapy be separated from x-ray diagnosis, and treatment and results in cancer of the breast.

HEART ailments and cancer were the No. 1 and 2 killers in the line-up of fatal diseases last year. Together they accounted for nearly 72 per cent of the deaths among American men and women. This was the experience of The Mutual Life Insurance Company of New York, according to a study made public yesterday of the causes of death among its 1,000,000 policy holders in 1947. Diseases of the heart and circulatory system caused more than 57 per cent of the deaths in all age groups combined but hit hardest among people over sixty years of age who accounted for nearly 62 per cent of deaths. Cancer, on the other hand, was most severely felt in the age group of forty to fifty-nine in which cancer caused about 16 per cent of deaths. Mortality from cancer in all age groups combined was 14.6 per cent of the total deaths from all causes. Accidents ranked third among the killers. The heaviest toll was among those under forty years of age in which accidents were the principal cause of death and accounted for nearly 36 per cent of all deaths in this age group. Suicide and tuberculosis also hit youth harder than they did the older age groups.



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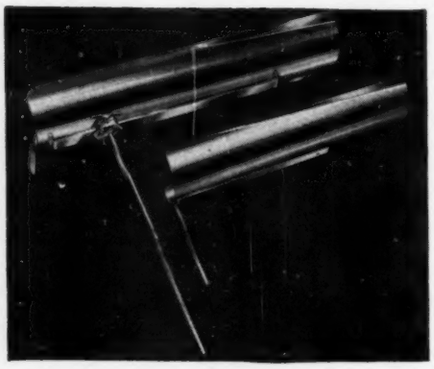

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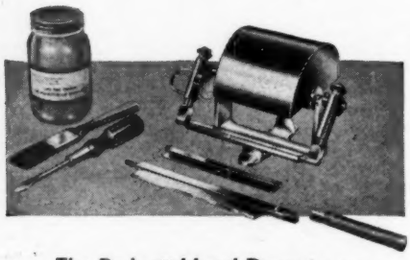

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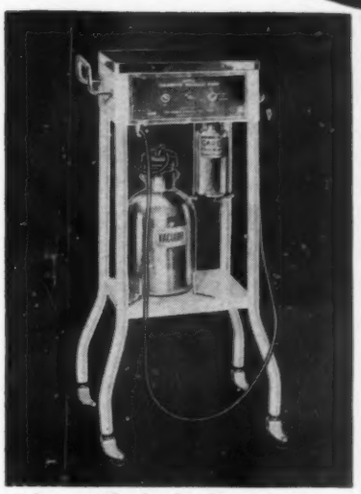

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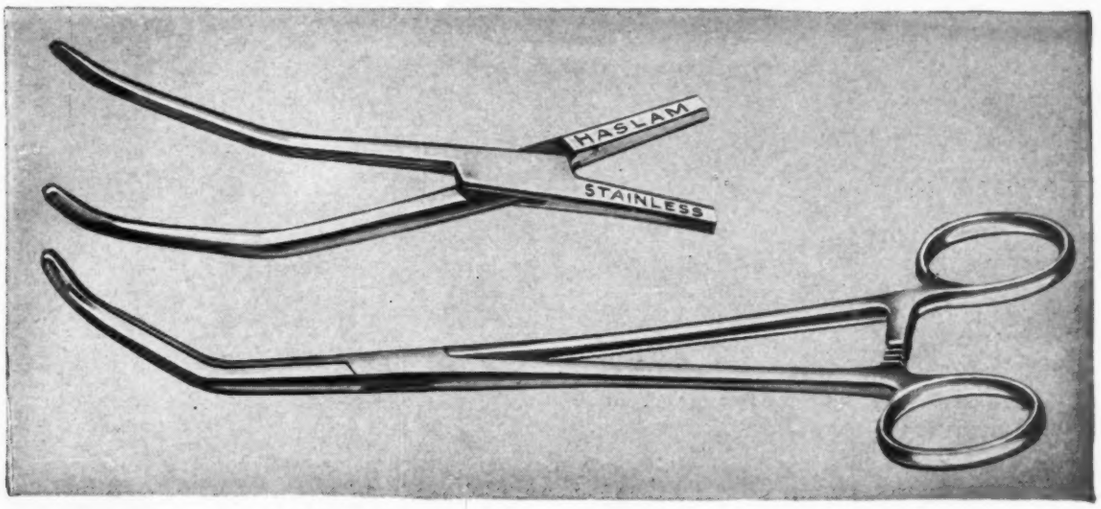

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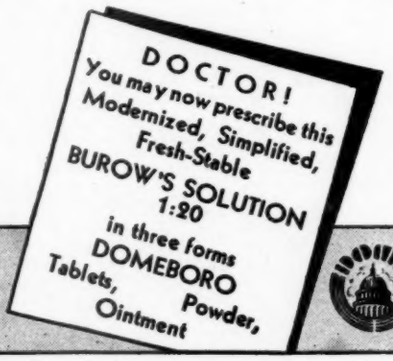

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The table indicates the nutrient values of this basic breakfast and the contribution made by 1 ounce of ready-to-eat or hot cereal* (whole grain, enriched, or restored to whole grain values of thiamine, niacin and iron), 4 ounces of milk and 1 teaspoonful of sugar.

The presence of this seal indicates that all nutritional statements in this advertisement have been found acceptable by the Council on Foods and Nutrition of the American Medical Association.

BASIC BREAKFAST	TOTALS supplied by Basic Breakfast		AMOUNTS supplied by cereal serving
Orange juice, 4 oz.;	CALORIES	611	202
Ready-to-eat or Hot Cereal, 1 oz.;	PROTEIN	20.7 Gm.	7.1 Gm.
Whole Milk, 4 oz.;	CALCIUM	0.465 Gm.	0.156 Gm.
Sugar, 1 teaspoon;	PHOSPHORUS	488 mg.	206 mg.
Toast (enriched, white), 2 slices;	IRON	3 mg.	1.6 mg.
Butter, 5 Gm. (about 1 teaspoon);	VITAMIN A	1074 I.U.	193 I.U.
Whole Milk, 8 oz.	THIAMINE	0.52 mg.	0.17 mg.
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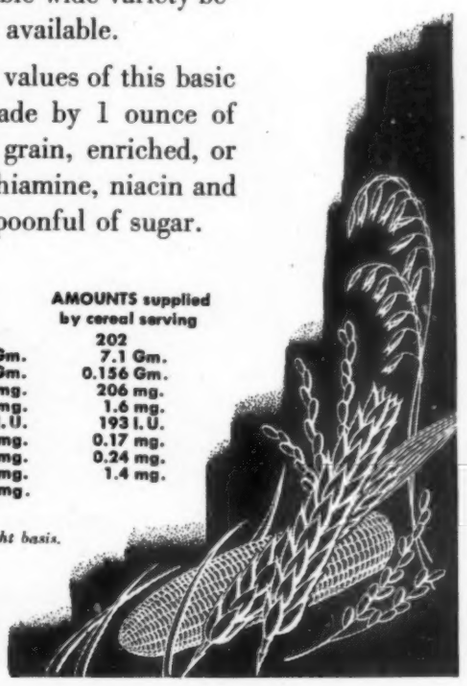
	NIACIN	2.3 mg.	1.4 mg.
	ASCORBIC ACID	64.8 mg.	

**Composite average of all breakfast cereals on dry weight basis.*



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